Divided Visual Field Study of Depression, Cognition, and Mood

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Abstract

This study compared the performance of 27 previously depressed and 21 never-depressed participants on a divided visual field task designed to examine the lateralization of emotional processing. Participants were asked to make judgments of emotional valence (positive or negative) for laterally presented words preceded by a centrally presented prime. Previous studies using this paradigm have found support for an enduring negativistic verbal processing bias in the right hemisphere among both depressed and previously depressed individuals. The present study represents an attempt to address several remaining questions regarding this phenomenon; specifically, this study aims to help clarify the respective roles of present mood state, anxious symptomatology, and prior depressive experience in giving rise to the aforementioned verbal processing bias. Participants in both diagnostic groups demonstrated an unusually large bias for words presented to the left hemisphere, such that their ability to accurately judge words presented to the right hemisphere was seriously compromised. Consequently, all participants who evidenced accuracy judgments for right hemisphere-presented words that were not significantly better than chance were excluded from further divided visual field analyses. The remaining 23 participants demonstrated a right visual field (left hemisphere) advantage for all words, and a valence-priming advantage for negative words. There were no significant differences between diagnostic groups, and no significant findings related to mood state for any of the divided visual field study variables, however, this was not unexpected given the limited power associated with this smaller sample of

participants. A number of hypotheses to account for the poor accuracy rates on this task – particularly for the right hemisphere – are discussed. Additionally, there was some evidence to suggest that the sad mood state associated with the mood induction may not have endured for participants, and implications for future research are discussed.

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Divided Visual Field Study of Depression, Cognition, and Mood

Approximately 20 million American adults experience depressive illness each year (Young, Weinberger, Beck, 2001). Because as many as 80% of previously depressed individuals will eventually relapse (Judd, 1997), considerable research attention has been accorded to the identification of factors that confer heightened vulnerability to depression recurrence. Moreover, research that increases our understanding of cognitive vulnerability in depression should provide new insights for diagnosis and treatment of depression. Therefore, this research is not only of theoretical importance for a better understanding of cognition in depression, but it could also have significant practical implications in the future.

Cognitive vulnerability to depression

Cognitive theories have enhanced our understanding of depression by identifying a potential mechanism for vulnerability. Such theories stress the etiological role of dysfunctional cognitions (e.g., negative beliefs and information processing patterns) in the development and maintenance of depression (Beck, 1976, 1987). Beck's influential cognitive model emphasizes the role of *schemas*, conceptualized as cognitive structures containing information stored in memory. Schemas function as cognitive templates that facilitate the screening and evaluation of incoming information. Although everyone has schemas, Beck suggests that those of depressed individuals are rigid and negative. Specifically, Beck's cognitive model proposes that depression is characterized by dysfunctional beliefs (schemas) about the self, the world, and the future – a constellation referred to as the "cognitive triad"

(Beck, 1970). The depressed person's schemas tend to reflect a view of the self as worthless, the world as filled with obstacles that are overwhelming and impossible to overcome, and the future as hopeless (Beck & Clark, 1988). These dysfunctional beliefs, in turn, bring about pervasive information processing biases in depression that encompass perception, attention, and memory.

Before we examine the empirical support for the proposed etiological role of dysfunctional cognitions (e.g., schemas, information processing patterns) in depression, it may be useful to consider a taxonomy proposed by Ingram and Kendall (1986) for organizing and understanding various cognitive constructs. According to this taxonomy, cognitive variables may be categorized as the *structure, proposition, operation,* or *product* of the information-processing system. The *structure* refers to the organization of internally stored information, comprised of the *propositions* or content (e.g., "I'm worthless."). Taken together, the *structure* and *propositions* make up the schema described by Beck (and, therefore, direct screening, encoding, and evaluation of information). *Operations* refer to the actual cognitive processes responsible for screening and encoding incoming information, as well as the retrieval of memories (e.g., information processes such as attentional and memory processes). Finally, *products* are the thoughts, decisions, and images that arise from the interaction of schemas with incoming information that is processed (e.g., automatic thoughts such as, "I must be stupid because I failed my exam").

Empirical support for the existence of dysfunctional beliefs (propositions or schematic content) and automatic thoughts (products) in depression is strong among

currently depressed individuals (reviewed in Haaga, Dyck & Ernst, 1991). Furthermore, depressed individuals generally process information (operations) in the environment with a negative bias by selectively attending to and remembering negatively toned information (reviewed in Gotlib & Neubauer, 2000; Haaga et al.). The evidence for a *causal* role of dysfunctional cognitions (e.g., propositions, operations, or products) in depression, however, is less clear (reviewed in Barnett & Gotlib, 1988; Ilardi & Craighead, 1999). For example, many researchers have argued that if dysfunctional cognitions play a causal role in the development of depression, such cognitions should be detectable (at some level of analysis) in previously depressed individuals, who are typically quite vulnerable to the experience of future episodes (Judd, 1997; Ingram, Miranda, & Segal, 1998). However, frequently utilized techniques for assessing dysfunctional beliefs (e.g., self-report measures such as the Dysfunctional Attitudes Scale, DAS, Weissman & Beck, 1988), and automatic thoughts (e.g., Automatic Thoughts Questionnaire, Hollon & Kendall, 1980) often fail to detect these dysfunctional cognitions in previously depressed, euthymic individuals (reviewed in Barnett & Gotlib, 1988; Haaga et al., 1991; Ilardi & Craighead, 1999). Furthermore, research that looks at information processing patterns in depression does not usually provide evidence for dysfunctional information processing patterns (e.g., operations such as memory or attentional biases for negative information) in previously depressed individuals in the absence of a sad mood (reviewed in Gotlib & Neubauer, 2000). The inability to detect dysfunctional cognitions (at any level of cognitive analysis) in previously depressed individuals in

the absence of a sad mood, however, does not prove that dysfunctional cognitions lack a causal role in the development and maintenance of depression. In fact, Beck (1987) has reemphasized that the cognitive model of depression suggests dysfunctional cognitions (particularly core beliefs or propositions) interact with stress in depression and, therefore, these cognitions may be latent until activated by a stressful life event.

Mood Priming and Cognitive Reactivity

Consistent with Beck's reemphasis on the interactive nature of dysfunctional cognitions and stress in depression, several researchers have examined the relationship between depressive cognition and current mood state, guided by the premise that maladaptive schemas (propositions) remain *latent* until reactivated by negative life events (e.g., Beck, 1987; Miranda & Persons, 1988). According to this *cognitive reactivity* model, dysfunctional cognitions (e.g., propositions, operations, and products) should be undetectable in vulnerable individuals until activated by stress that leads to a depressed mood or a temporary sad mood that increases the accessibility of these cognitions (Miranda & Persons; Ingram, Miranda, & Segal, 1998). Studies that have investigated the cognitive reactivity model have typically employed mood induction procedures to bring about a temporary sad mood, and have generally provided support for it (e.g., Ingram, Bernet & McLaughlin, 1994; Williams, 1988, as cited in Ingram et al., 1998; Segal, Gemar, & Williams, 1999). These studies mostly find that previously depressed, while in a sad mood state, individuals exhibit dysfunctional cognitions (e.g., beliefs/propositions, information

processing patterns or operations) that are not observed among never-depressed individuals in a sad mood. Notably, however, a few studies have not provided support for the cognitive reactivity hypothesis (e.g., Blackburn & Smyth, 1985; Dykman, 1997, as cited in Ingram et al., 1998; Gotlib & Cane, 1987).

Studies investigating the cognitive reactivity model for depression usually assess dysfunctional cognitions in previously depressed and never-depressed individuals before sad mood priming and then reassess dysfunctional cognitions following mood priming procedures. For example, in a study by Teasdale and Dent (1987), the authors initially found no reliable differences between previously depressed and never-depressed participants on an incidental recall task of self-referent adjectives (a task that likely assesses cognitive propositions and operations). Following a sad mood induction in which a temporary sad mood was primed, however, previously depressed participants recalled more negative adjectives that they had endorsed as self-descriptive compared with never-depressed participants. Thus, prior to mood priming, the recall performance of previously depressed participants was indistinguishable from never-depressed participants, and only after mood priming did they exhibit dysfunctional cognitions (information processing patterns reflective of propositions) consistent with depression.

Miranda and Persons (1988) also examined the cognitive reactivity hypothesis using previously depressed and never-depressed participants. Previously depressed participants did not initially report significantly elevated dysfunctional attitudes on the DAS (the DAS is usually regarded as a measure that taps into deeper cognitive

constructs such as propositions) compared with never-depressed participants. Following a sad mood priming procedure, however, previously depressed participants endorsed dysfunctional attitudes, whereas never-depressed participants did not. Moreover, in a subsequent study, Miranda, Persons and Byers (1990) examined the relationship between current mood state and dysfunctional attitudes (using the DAS) of previously depressed and never-depressed participants by looking at naturally occurring diurnal variations in mood. They found that previously depressed participants had elevated DAS scores while in a naturally occurring negative mood state; DAS scores of never-depressed participants were not significantly related to diurnal variations in mood (Miranda et al., 1990). Finally, Miranda, Gross, Persons, and Hahn (1998) obtained similar results, whereby previously depressed participants endorsed dysfunctional attitudes on the DAS following an induced temporary sad mood state that increased with the intensity of their sad mood. Negative mood, however, was not associated with increased scores on the DAS in never-depressed participants (Miranda et al., 1998).

In a study by Ingram and colleagues (1994), information processing patterns were examined in previously depressed and never-depressed participants. All participants were first randomly assigned to either a control condition or a mood induction condition. Immediately following the mood induction and control condition tasks, participants performed a dichotic listening task to assess attentional allocation (information processing patterns influenced by schematic content). The task required participants to attend to a story that was presented in one ear while

simultaneously ignoring distractor words presented to the other ear; distractor words were positive and negative adjectives, and neutral nouns. Errors for tracking the story served as the dependent variable used to assess attentional allocation (more errors reflected greater attention to distractor words). The authors found no significant differences in attentional allocation between previously depressed and never-depressed participants in the control condition. For participants primed for a temporary sad mood state, however, previously depressed participants evinced greater attentional allocation to both positive and negative stimuli, relative to neutral stimuli as compared with never-depressed participants in the sad mood condition (and compared with both groups of participants in the control condition). Ingram and Ritter (1998) used the same dichotic listening task in a subsequent study and found that previously depressed participants were indistinguishable from never-depressed participants prior to mood priming. Following sad mood priming, however, previously depressed participants had increased errors for tracking the story when negative words were presented to the other ear. Thus, unlike the findings from the earlier study, previously depressed participants showed an attentional bias that was specific to negative stimuli rather than emotional stimuli in general (Ingram & Ritter, 1998).

Hedlund and Rude (1995) examined the relationship between dysfunctional cognitions and depression vulnerability with the emotional Stroop task, a scrambled sentence task, and an incidental recall task following a self-focus induction procedure intended to prime a negative mood. The emotional Stroop task is a modified version

of the original Stroop task, and uses adjectives associated with different emotional states (e.g, happy, sad). For this task, participants are presented with words written in different colors, and they are required to name the color of each word (not the actual word) as quickly as possible. Slowed naming of colors for specific types of words (e.g., depression-related) is believed to be a result of increased attention for those words and a resulting interference that leads to slower color-naming. The authors found that in the presence of self-focused attention, there were no significant differences between currently depressed, previously depressed, and never-depressed participants on the Stroop test. On the scrambled sentences task, however, currently and previously depressed participants completed significantly more negative sentences, and they recalled more negative self-descriptive adjectives on the incidental recall task compared with never-depressed participants (following the self-focus induction). Currently depressed participants completed significantly more negative sentences than previously depressed participants in the presence of self-focused attention, but they were not reliably different from previously depressed participants on the recall task (Hedlund & Rude). Thus, following a self-focused mood induction procedure, previously depressed participants did not reliably differ from currently depressed participants on tasks assessing patterns of information processing that are likely influenced by dysfunctional schemas (i.e., a recall task).

Finally, Smith, Teasdale, and Cowen (1998; cited in Ingram et al., 1998), used a tryptophan depletion paradigm to create a biologically-induced sad mood in previously depressed and never-depressed women. Previously depressed participants

who experienced a more severe drop in mood demonstrated more positive responses to measures of dysfunctional cognitions (DAS, Attributional Style Questionnaire, and an autobiographical memory test) than previously depressed participants who experienced a less severe drop in mood. Moreover, previously depressed participants who experienced a more severe drop in mood also demonstrated significantly more positive cognitive responses on some measures than never-depressed participants in the presence of a biologically-induced sad mood. The authors explained that previously depressed participants may attempt to consciously control their thinking when they experience strong shifts in mood in order to compensate for low affect, whereas never-depressed participants may not be as motivated to consciously control their thinking, however, additional studies are needed to determine whether this finding is reliable (Teasdale et al.; as cited in Ingram et al., 1998). Furthermore, the measures used in this study may be more amenable to conscious control than tests of information processing patterns, which examine cognitive processes that are fast and outside of conscious awareness (e.g., attentional processes). Thus, it's possible that the results obtained for this study would have been different if the dysfunctional cognitions examined were fast and unconscious information processes.

The great majority of studies reviewed herein are at least consistent with the cognitive reactivity hypothesis for depression. Furthermore, these studies stand in stark contrast to the null results found in many studies (e.g., reviewed in Haaga et al., 1991) that failed to identify dysfunctional cognitions among previously depressed participants in the absence of negative mood induction. The strongest support for a

causal role for dysfunctional cognitions (propositions, operations, and products) in depression, however, comes from longitudinal studies of the cognitive reactivity hypothesis. For example, Williams (1988) investigated depression vulnerability in undergraduate students and found that participants who recalled more negative (than positive) self-referent adjectives in response to a sad mood state were more likely to become depressed one year later. The results suggest that cognitive reactivity in response to a sad mood may predict the development of a future depressive episode. In another longitudinal study by Beevers and Carver (2003), undergraduate students were recruited with varying histories of major depression. Current levels of dysphoria were assessed in all participants prior to performing a dot-probe task designed to assess attentional allocation. For this task, pairs of words that were either positive or negative were briefly presented on a computer screen and then were subsequently followed by a dot in the spatial location of one of the words. Participants were asked to indicate as quickly as possible the location of the dot probe. Faster detection of the probe for specific classes of stimuli is interpreted as reflecting an attentional bias for such stimuli. After completing the dot probe task, participants underwent a negative mood induction and then performed the dot probe task again. At the follow-up session, participants completed a measure of intervening life stress, and level of dysphoria was also assessed. The authors found that greater attention to negative stimuli on the dot probe task (i.e., faster detection of probes that followed negative words) following sad mood priming interacted with life stress to predict increases in dysphoria two months later.

Finally, in a longitudinal study that spanned several years, Segal and colleagues (1999) recruited previously depressed individuals who had undergone either cognitive therapy or pharmacotherapy during their most recent episode of depression. The authors found that depression treatment modality (cognitive therapy or medication) predicted cognitive reactivity to a temporary sad mood state. Participants who received pharmacotherapy endorsed and increased number of dysfunctional attitudes (assessed with the DAS) following mood priming, whereas those participants who received cognitive therapy did not generally demonstrate cognitive reactivity. Furthermore, this cognitive reactivity (regardless of the type of treatment) was predictive of depression relapse several years later; participants who did not respond with dysfunctional attitudes following negative mood priming were significantly less likely to relapse compared with participants who endorsed dysfunctional attitudes following the sad mood induction. Thus, research exploring mood priming and cognitive reactivity suggests the existence of latent dysfunctional cognitions (e.g., propositions, operations, products) in depression-vulnerable individuals (e.g., previously depressed individuals and individuals who go on to develop a major depressive episode) that are uncovered during a sad mood state.

The cognitive reactivity literature generally provides at least partial support for a causal role of dysfunctional cognitions in the development and maintenance of depression, in as much as depression-vulnerable individuals appear to respond differently to a sad mood than do individuals who are not vulnerable to depression (e.g., individuals who have never been depressed). The longitudinal studies (Beevers

& Carver, 2003; Segal et al.; Williams, 1998) provide considerable support for a causal role of dysfunctional cognitions in the development of future depressive episodes in vulnerable individuals because cognitive reactivity was associated with subsequent development of depression (or dysphoria) in all three studies. Many of the studies investigating the cognitive reactivity model, however, are not without methodological limitations, which raise questions regarding their implications for cognitive theories of depression. For example, with the exception of a few studies (e.g., Beevers & Carver; Ingram et al., 1994; Segal et al., 1999), most investigations of the cognitive reactivity model assessed mood only immediately after mood priming procedures, and did not reassess mood at any other point to ensure that the sad mood was of equal duration among both groups of participants (previously depressed and never-depressed). Thus, even though previously depressed and never-depressed participants achieved an equally sad mood immediately following mood priming procedures, it remains possible that the depressed mood was not equally maintained by both groups. As a result, previously depressed participants may have demonstrated greater dysfunctional cognitions because of a more persistent sad mood rather than latent dysfunctional cognitions that emerged during a sad mood. Accordingly, future studies investigating the cognitive reactivity hypothesis and depression vulnerability should include an assessment of mood following administration of dysfunctional cognition measures (e.g., the DAS) and/or performance on a cognitive task designed to evaluate information processing patterns.

Another methodological limitation associated with the cognitive reactivity literature is the fact that comorbid anxiety has not typically been assessed. Because anxiety has a high rate of comorbidity with depression (American Psychiatric Association, 2000), research that has examined the cognitions of either depressed or anxious participants has likely included participants with a high degree of symptomatology associated with both disorders (or states). Thus, in reality, many of the research findings on dysfunctional cognitions (propositions, operations, and products) in depression and anxiety may reflect dysfunctional cognitions associated with both disorders or states rather than dysfunctional cognitions associated with "pure" depression or anxiety. Therefore, it is important for studies to assess the degree of anxious and depressive symptoms in their participants so that it is clear as to whether any dysfunctional cognitions found, reflect those associated with depression, anxiety, or both disorders (or states). Moreover, studies that have explicitly examined dysfunctional cognitions (propositions, operations, products) in individuals with depression and co-occurring anxiety have produced more complex results (e.g., Clark, Beck, & Stewart, 1990; Ingram, Kendall, Smith, Donnell & Ronan, 1987). For example, although individuals with "pure" anxiety have not consistently demonstrated negative automatic thoughts (e.g., "I must be stupid because I failed my exam"), individuals with comorbid depression and anxiety often demonstrate relatively more of these thoughts than individuals with a "pure" depression, suggesting a complex interaction between depression and anxiety (e.g., Clark et al., 1990; Ingram et al., 1987). Accordingly, the current project will assess

for symptoms of anxiety in addition to depression to determine whether findings reflect dysfunctional cognitions associated with depression, anxiety, or only for comorbid anxiety and depression.

Finally, findings consistent with the cognitive reactivity model do not preclude dysfunctional cognitions from being present in depression-vulnerable individuals in the absence of a sad mood state. In fact, it remains possible that dysfunctional cognitions at one level of analysis (or one type of cognitive construct) remain detectable even in the absence of depressed mood. Furthermore, if these cognitive constructs (e.g., propositions, operations, and products) are latent in vulnerable individuals when they are not depressed, it is reasonable to hypothesize that such constructs must be encoded *somewhere* within the brains of these individuals, even in the absence of a sad or depressed mood. In this regard, a recent study by Atchley, Ilardi, and Enloe (2003) used a cognitive neuroscience approach (divided visual field) to study cognition in depression, and found evidence for dysfunctional cognitions (information processing patterns or operations) among previously depressed individuals in the absence of a sad mood state. Moreover, a replication study by Atchley, Stringer, Mathias, Ilardi, & Minatrea (2005) produced similar findings, whereby previously depressed participants demonstrated dysfunctional information processing patterns with the divided visual field method in the absence of a sad or depressed mood. Thus, the studies by Atchley and colleagues suggest that latent dysfunctional cognitions (in this case information processing

patterns) can be detected in vulnerable individuals in the absence of a depressed mood using cognitive neuroscience methods.

Cognitive Neuroscience Approach

The cognitive neuroscience (CNS) perspective has been used to study how brain events give rise to various mental and behavioral events. In this regard, the CNS approach has been used to investigate emotion by examining the relationship between brain activities and the way people process emotional stimuli and experience different emotional states. Relevant to the current proposed study, there are two related but largely distinct lines of inquiry that have used the CNS perspective to study emotion, and have put forth different hypotheses regarding the neuropsychology of emotion. The first, which is referred to as the right hemisphere (RH) dominance hypothesis, has focused on the processing and comprehension of emotional stimuli, and has found the right hemisphere to be superior to the left hemisphere in the processing and comprehension of emotional stimuli (e.g., Etcoff, 1984; 1989; Tucker, Watson & Heilman, 1977). Many of the studies providing support for this theory have evaluated individuals with cortical damage, and have found that right hemispheric damage typically impairs processing and comprehension of emotional stimuli such as facial expressions and tone of voice . In contrast, left hemispheric damage generally does not impair these functions (Etcoff; Tucker et al.). Furthermore, when stimuli are presented in isolation to the cerebral hemispheres (e.g., using divided visual field or dichotic listening methodology), participants without brain damage generally demonstrate enhanced processing (i.e., greater accuracy and

speed) of emotional stimuli when presented to their right versus left hemisphere (e.g., King & Kimura, 1972; Ley & Bryden, 1982; Strauss & Moscovitch, 1981).

The second area of research using the CNS approach to study emotion involves the examination of hemispheric differences in the experience of various emotional states, and has provided support for the valence hypothesis of emotion. In this line of inquiry, the left prefrontal cortex appears to play a role in mediating the experience of positive emotions (e.g., euphoria) and approach-related behaviors (e.g., introducing yourself to a stranger), whereas the right prefrontal cortex appears to be important in the experience of negative emotions and withdrawal-related behaviors (e.g., avoiding an aversive stimulus; Davidson & Tomarken, 1989; Henriques & Davidson, 1991; Tucker et al., 1981). Moreover, these lateralization effects (i.e., differences between the left and right hemisphere) have been found both in nonclinical populations and in individuals with mood disorders. Specifically, individuals who have experienced negative affect or depression show greater right prefrontal cortex activation relative to the left prefrontal cortex as compared with euthymic, non-depressed individuals (Davidson & Tomarken; Henriques & Davidson). Notably, this asymmetry between the hemispheres in depressed and temporarily sad individuals seems to be the result of reduced activation of the left prefrontal cortex rather than increased activity in the right prefrontal cortex (Davidson, 2004; Henriques & Davidson, 1991; Shagass, 1972).

A third theory on the neuropsychology of emotion has sought to integrate the two aforementioned hypotheses and the research associated with each. The

circumplex model of emotion was originally described by Russell (1980) and suggested that affect is organized based on two orthogonal dimensions (or 2 dimensions in space), which are pleasure vs. displeasure (valence) and degree of arousal. Heller (1993) subsequently attempted to integrate this conceptualization of emotion with neuropsychological findings on emotion. According to Heller, the posterior right hemisphere is responsible for perceiving all emotion and modulating autonomic and behavioral arousal in emotion, whereas the anterior regions are significant to the experience of emotional valence (i.e., left frontal region is implicated in the experience of positive emotional states, and the right frontal region is implicated in negative emotional states). Notably, Heller's circumplex model of emotion is consistent with research supporting the right hemisphere dominance and valence hypotheses of emotion (e.g., Davidson & Tomarken, 1989; Etcoff, 1984; 1989; Henriques & Davidson, 1991; Tucker, Watson & Heilman, 1977; Tucker et al., 1981). Thus, according to Heller's model, the experience of depression would be associated with greater right anterior activation relative to left anterior activation as was discussed within the valence hypothesis of emotion. Furthermore, Heller's model suggests that anxious states (or anxiety) would be associated with greater posterior right hemisphere activation (reflecting greater arousal) relative to non-anxious states (or individuals with relatively less anxiety), which has been supported by numerous research findings (e.g., Bruder et al., 1997; Davidson, 1998; Heller, Nitschke, Etienne, Miller, 1997). Thus, given that anxiety is frequently comorbid with depression, and that specific (and distinct) patterns of hemispheric activation are

associated with depressive and anxious states (and disorders), studies investigating information processing patterns in depression should assess for possible anxiety (heightened anxious states or anxiety disorders) to determine whether findings are specific to depression, or if they are associated with comorbid depression and anxiety.

Significance of the CNS Approach to the Study of Cognitive Vulnerability in Depression

Unlike traditional methods for studying emotion and mood disorders, the CNS perspective is distinctive for using objective measures of emotional processing and experience (e.g., brain activation, hemispheric differences, etc.). Thus, the CNS approach has the ability to explore brain activity and cognitive processes for which the individual is largely unaware. Given the high rate of depression recurrence (Judd, 1997) and the possibility that dysfunctional cognitions (at some level of cognitive analysis) exist in vulnerable individuals outside of the depressed state - but remain hidden and difficult to detect - this approach remains a potentially viable way to tap latent dysfunctional cognitions that might render these individuals vulnerable to future depressive episodes.

In this regard, the aforementioned study by Atchley and colleagues (2003) utilized CNS methods (divided visual field presentation) to investigate information processing patterns (operations) in currently depressed, previously depressed, and never-depressed individuals. The divided visual field presentation possesses an advantage over central visual field presentation (used in most studies on information processing in depression) because it assesses functioning of the left and right

hemispheres separately, and therefore reduces the possibility of subtle effects within each hemisphere "washing out" combined effects for both hemispheres together. Participants in this study viewed negative or positive adjectives that were presented to either the left or right visual field (right or left hemisphere, respectively), and they were asked to indicate the valence of the adjective (negative or positive). Using the divided visual field method, Atchley and colleagues found evidence for a right hemispheric processing advantage for negatively valent self-descriptive adjectives among depressed and previously depressed participants (i.e., they showed greater accuracy and faster reaction times for detecting negative adjectives compared to positive adjectives when presented to the left visual field/right hemisphere) – a pattern not observed in never-depressed participants. Moreover, in a subsequent study by Atchley and colleagues (2005), similar results were obtained; depressed and previously depressed participants demonstrated greater accuracy (reaction time was not analyzed) for negative words (compared with positive words) presented to the left visual field (right hemisphere). Thus, unlike studies using self-report measures to assess dysfunctional beliefs (e.g., propositions, reviewed in Haaga et al., 1991), or central visual field presentation of stimuli (reviewed in Gotlib & Neubauer, 2000) to assess information processing patterns, Atchley and colleagues found evidence for dysfunctional information processing patterns in previously depressed individuals in the absence of an induced sad mood by investigating these processes in the hemispheres separately.

Atchley and colleagues (2003, 2005) interpreted their findings as consistent with the hypothesis that semantic networks (i.e., networks containing information about the meanings of words) associated with the right hemisphere are organized by emotional experience (e.g., depression-experience) because depression-experienced participants evidenced facilitated processing (faster response time) of negative words when they were preceded by a negative prime word compared with when negative words were preceded by a positive prime word (i.e., an advantage when the prime and target matched for valence). The significance of this later finding to right hemisphere semantic structure is that it suggests a faster spread of activation for negatively valent word meanings – compared with positively valent word meanings - within the right hemisphere for depression-experienced individuals. With a greater developed semantic network for negative word meanings in the right hemisphere, and therefore faster spreading of activation, related negative word meanings would become readily available much faster for depression-experienced individuals. Moreover, because persistent right hemisphere biases for the processing of negatively valent emotional words were found in previously depressed participants (in the absence of an induced sad mood), the right hemisphere semantic networks may reflect an enduring marker of depression vulnerability in previously depressed individuals.

However, even though Atchley and colleagues were able to detect dysfunctional information processing patterns in previously depressed individuals in the absence of an induced sad mood, their studies are not without methodological limitations that raise questions regarding the interpretation of their results. One of the

major limitations is the fact that anxiety was not assessed in either study. This is significant given that anxiety frequently co-occurs with depression, and detection of dysfunctional cognitions varies depending on whether participants are diagnosed with depression, anxiety, or co-morbid depression and anxiety (e.g., Clark et al., 1990; Ingram et al., 1987). Furthermore, other studies that have investigated lateralization (differences between the hemispheres) of information processing patterns in depressed and comorbid depressed and anxious individuals have not always found reliable differences between individuals with comorbid depression and anxiety and never-depressed, non-anxious control participants (reviewed in Heller & Nitschke, 1998). Thus, it is important to determine whether the dysfunctional information processing patterns in previously depressed participants that were observed by Atchley and colleagues are specific to depression or are only detectable when depression and anxiety co-occur.

Another major limitation associated with the studies of Atchley and colleagues is the fact that current mood state was not assessed (the relative absence of dysphoria among previously depressed participants was inferred on the basis of low scores on the Beck Depression Inventory). Although the authors interpreted their findings as consistent with a stable, enduring bias for negative information in depression-vulnerable individuals (e.g., previously depressed individuals), unaffected by current mood state, since current mood state was not explicitly assessed, it remains possible that previously depressed participants were in a sad mood at the time they performed the divided visual field task. If so, then the observed bias for negative

information (i.e., greater accuracy for information presented to the right hemisphere) may have been state-dependent (i.e., the bias was a result of their sad mood) rather than reflecting an enduring negative bias that persists in the absence of a sad mood state. Moreover, because Atchley and colleagues conducted the diagnostic interview at the beginning of their procedure (prior to the divided visual field task), it remains a viable possibility that previously depressed (and currently depressed) participants were inadvertently *primed* for a sad, negative mood because they had experiences with depression that they were discussing with the interviewer. Never-depressed participants, however, had fewer experiences with depressive symptoms to report, and therefore, would not likely have been primed for a sad mood by the clinical interview. Thus, if previously depressed participants were experiencing a sad/depressed mood at the time they performed the divided visual field task, then Atchley and colleagues' findings could be interpreted as reflecting a state-dependent bias for negatively valent information (rather than a persistent negative bias) that was found in depression-vulnerable individuals. Alternatively, if previously depressed and never-depressed participants were both in a sad mood when they performed the divided visual field task (which is a possibility because mood was not assessed), then the findings may be consistent with the cognitive reactivity hypothesis, whereby dysfunctional cognitions (in this case information processing patterns) were reactivated by a negative mood in depression-vulnerable individuals. Without an assessment of mood, however, it is impossible to discriminate between three possible interpretations of their findings (i.e., as an enduring negative bias in the absence of

sad mood, a state-dependent bias, or findings consistent with the cognitive reactivity hypothesis). Thus, the current project will combine mood priming techniques and assessment of current mood state with the divided visual field method to help differentiate between these three possible interpretations of Atchley and colleagues' findings.

Summary and Relevance to the Present Study

The research reviewed herein clearly documents the occurrence of dysfunctional cognitions in currently depressed individuals (reviewed in Haaga et al., 1991 and Gotlib & Neubauer, 2000). The evidence for dysfunctional cognitions in previously depressed individuals in the absence of a sad mood, or in individuals with varying degrees of anxious symptoms and depression, is less clear. Preliminary research, however, suggests that by using research methodology that investigates cognitions in the hemispheres separately (e.g., divided visual field), it may be possible to detect latent dysfunctional information processing patterns in previously depressed individuals in the absence of an induced sad mood (Atchley et al., 2003; 2005). This finding of an apparent enduring bias for negative information (compared with positive information) only in the right hemisphere for previously depressed individuals is significant because previous research has failed to detect the presence of stable dysfunctional cognitions in depression in the absence of an induced sad mood (reviewed in Ingram et al., 1998). To date, however, there are no published studies that have used the divided visual field method to study the effects of a primed sad mood on cognitions in previously depressed and never-depressed individuals,

which may prove useful for determining the significance of mood and cognitions to depression vulnerability. Thus, the aim of the present study was to combine mood priming with the divided visual field method to tease apart the effects of a sad mood state and depression-experience on cognition. Moreover, by including an assessment of anxiety-related symptoms, this study was designed to explore possible differences in the lateralization of information processing patterns between individuals with a more "pure" depression and individuals with depression and comorbid anxious symptoms using the divided visual field methodology.

Although the methodology of the present study was not capable of directly resolving the question of whether dysfunctional cognitions *cause* depression or vice versa, it was designed to provide evidence of considerable relevance to this question by examining the interaction between current mood, depression experience, and anxious symptoms using what may be a more sensitive methodology (divided visual field as opposed to central visual field presentation) for detecting dysfunctional information processing patterns. For example, if the results showed that never-depressed individuals in a sad mood exhibit a right hemispheric bias for negative material similar to that of currently and previously depressed individuals, this would suggest that there is something about a sad mood itself that leads to dysfunctional information processing patterns (i.e., anyone in a sad mood would experience some dysfunctional cognitions). If this were the case, previously depressed individuals may evidence more depressotypic cognition simply because they experience sad moods more often. If, however, the results showed that sad, never-depressed

individuals do not demonstrate a preference for negative material in their right hemisphere comparable with that of depression-experienced individuals, then the findings would be consistent with Beck's model, which emphasizes a cognitive vulnerability in depression.

Furthermore, if never-depressed and previously depressed participants' performance on the divided visual field task was not significantly affected by a temporary sad mood (and their mood is not sad during their performance on the task prior to mood priming), then this would suggest that Atchley and colleagues' interpretation of their findings was accurate. Thus, the findings would provide robust evidence for a stable, enduring negative bias in the right hemisphere of depression-vulnerable individuals that is not state-dependent. Moreover, the findings would provide further support for the hypothesis that the right hemisphere semantic networks are organized by emotional experience (because a sad mood was not necessary to detect a bias for negative words in the right hemisphere of depression-vulnerable individuals), and the possibility that these networks are an enduring marker of depression vulnerability. Thus, by exploring the interaction between current mood and depression experience on the lateralization of information processing patterns, this study attempted to address an important theoretical question as to the significance of negative mood and cognition in depression vulnerability.

Study Hypotheses

Specific predictions for the proposed study were based on empirical findings and theory. Atchley and colleagues (2003, 2005) used a similar task and, therefore,

predictions regarding differences between diagnostic groups on the dependent variables are based on those empirical findings. Moreover, cognitive theories of depression suggest that depressed and depression-vulnerable individuals have greater access to negative information because they possess a well-developed network for negative information (i.e., strong associations for negative information resulting in more efficient processing of this information), which makes this information more accessible (e.g., Bower, 1981, 1987).

First, it was hypothesized that depression-experienced participants would exhibit dysfunctional information processing patterns (i.e., faster and more accurate processing of negative information compared with positive information) in the right hemisphere. Specifically, it was predicted that depression-experienced participants would be faster and more accurate at judging negatively valent adjectives relative to positively valent adjectives, particularly when negative adjectives were preceded by another negative adjective (valence-priming), and never-depressed participants would evidence the opposite pattern of results in the right hemisphere (i.e., faster and more accurate at judging positively valent adjectives, especially when preceded by another positively valent adjective). Second, it was predicted that sad mood priming would have no appreciable effect on information processing patterns in the right hemisphere for previously depressed or never-depressed participants (which would be consistent with Atchley and colleagues interpretation of their findings for a trait-like, enduring negative bias in depression vulnerable individuals). Finally, in light of the remaining questions surrounding the significance of co-morbid anxiety to lateralized

dysfunctional information processing patterns in depression, no specific predictions were made for co-occurring anxiety.

Method

Participants

Eighty-seven undergraduate students attending the University of Kansas were recruited for the study in fulfillment of meeting a course requirement. Participants were recruited based on pretest responses to questionnaires administered through a mass prescreening measure on-line. For the prescreen measures for this study, students completed the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996) to assess any current depressive symptoms and a brief questionnaire based on the diagnostic criteria enumerated in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM IV, American Psychiatric Association, 1994) for a current or previous Major Depressive Episode (MDE). Based on pretest responses to these questionnaires, 3 tentative groups of individuals were identified. Individuals who endorsed at least 5 core diagnostic criteria for a MDE and had a score of 18 or greater on the BDI-II were given a tentative diagnosis of "depressed." Individuals who indicated that they previously would have endorsed at least 5 core diagnostic criteria for a MDE (but not presently), and had a score of less than 9 on the BDI-II were tentatively categorized as "previously depressed." Individuals who endorsed 2 or fewer diagnostic criteria for a MDE for their lifetime and a score of 9 or less on the BDI-II received a tentative classification of "never depressed." Moreover, individuals recruited for the study were native English-speakers and right-handed (according to their responses on the prescreen measure). Screened individuals who

tentatively met all study criteria and met criteria for one of the aforementioned diagnostic groups were contacted by phone or email to participate.

Recruited participants subsequently completed a brief screening measure (Inventory to Diagnose Depression, Life time version, Zimmerman & Coryell, 1987) to provide a probable diagnosis of depressed, previously depressed or never-depressed. Participants given a probable diagnosis of never-depressed were randomly assigned to one of two groups. One of the never-depressed groups was treated like the previously depressed participants and underwent mood priming. The other never-depressed group of participants did not undergo mood priming. The purpose of the later group of participants was to make certain that the two word lists for the divided visual field task were equivalent, and that there were no significant fatigue or practice effects over time, and were not included in the primary study analyses. All participants were administered a clinical interview (Structured Clinical Interview for DSM-IV Axis I Disorders, clinician version, SCID; First, Spitzer, Gibbon & Williams, 1996) conducted by an advanced graduate student (the first author) in clinical psychology to confirm their depression status. The BDI-II was administered to assess depression symptom severity at the time of the study. All participants who were retained for the study never met criteria for a manic or hypomanic episode. Individuals who met 5 or more diagnostic criteria for a MDE, and had a score of 15 or higher on the BDI-II comprised the depressed group. Individuals who previously met 5 or more diagnostic criteria for a MDE but currently met 2 or fewer diagnostic criteria, and who had a score of 9 or less on the BDI-II

comprised the previously depressed group. Individuals who met 2 or fewer diagnostic criteria for a MDE (and never met 5 or more diagnostic criteria), and who had a score of 9 or less on the BDI-II comprised the never-depressed groups. Additionally, anxiety was assessed using the State-Trait Anxiety Inventory (STAI; Spielberger, 1968). Twelve study participants were eliminated from all analyses because they did not meet study criteria. Moreover, data for currently depressed participants was not analyzed because only 10 recruited study participants met criteria for this diagnostic group, and therefore, would have limited the power for detecting significant between (and within) subjects effects by including them in the analyses.

Measures

1. *Beck Depression Inventory-Revised (BDI-II).* A widely used self-report measure that assesses the severity of depressive symptoms for the last two weeks. The BDI-II consists of 21 items answered on a scale of 0 to 3, with totaled scores that range from 0 to 63. Higher scores indicate greater depression symptom severity. The BDI-II has high internal consistency, with alpha coefficients of .92 and .93 for psychiatric outpatients and college students respectively (Beck, Steer, & Brown, 1996). The BDI-II also has well-established validity for depression screening in clinical and non-clinical populations. Moreover, the BDI-II correlates highly with the Hamilton Psychiatric Rating Scale for Depression (HRSD; Hamilton, 1960), r = .71, which is another widely used measure to assess depressive symptoms (Beck et al., 1996).

2. *The Inventory to Diagnose Depression, Lifetime Version* (IDD-L; Zimmerman & Coryell, 1987). The IDD-L is a 22-item self-report inventory that assesses the level and duration of previous depressive symptomatology; scores of 40 and above are indicative of a previous depressive episode (Soloman, Haaga, Brody, Kirk, & Friedman, 1998). The IDD-L has comparable sensitivity and specificity to the Diagnostic Interview Schedule (Zimmmerman & Coryell) and good discriminant validity (Sakado, Sata, Uehara, Sato, & Kameda, 1996) and test-retest reliability (Sato et al., 1996) has been reported. Administration to college and community samples has yielded a Spearman Brown split-half reliability coefficient of .90 and a Cronbach alpha of .92 (Zimmmerman & Coryell).

3. *The Major Depressive Episode Screening Measure* (created and used by Atchley et al., 2003; 2005). It is based on the diagnostic criteria for a MDE as described in the DSM-IV (APA, 1994). Participants will be given a tentative classification for their projected diagnosis on the basis of this screening measure and the BDI-II. Reliability and validity of this brief screening measure is determined subsequently by diagnoses provided by the SCID for DSM-IV diagnoses, clinician version (First, et al., 1996).

4. *Multiple Affect Adjective Checklist-Revised* (MAACL-R; Zuckerman & Lubin, 1985). The MAACL-R was used in the present study to assess level of depressive affect within an experimental session. The MAACL-R contains 66 scorable adjectives, and participants are instructed to check each item that applies to how they feel at that moment. For the present study, depression subscale scores were

used to assess participants' negative affect following a sad mood-priming procedure. For the depression subscale, participants receive a point for each negative adjective they endorse and a point for each positive adjective that they do not endorse so that higher scores indicate greater depressive affect. The MAACL has well-established reliability and validity data for assessing depressive affect. The depression subscale of the MAACL has shown a split-half reliability coefficient of .92 (Zuckerman, Lubin, Vogel, & Valerius, 1964). Moreover, the depression subscale has been shown to correlate highly (r=.60) with adolescents' self-report ratings of sadness and depression that were based on a 5-point Likert scale (Lubin et al., 1986).

5. *Visual Analogue Scale* (VAS; Grossberg & Grant, 1978). Participants will rate their current mood on a VAS measuring 76 mm from center to each of two endpoints. The descriptor *not sad at all* is located to the left of center, and *very sad* is located on the right side, with an arrow indicating increasing strength of mood associated with greater distance from the center. Furthermore, the VAS for assessing mood has demonstrated reliability and validity (Ahearn, 1997). Ahearn and Carroll (1996) investigated individuals with major depression and found that they demonstrated a test-retest reliability coefficient of .92 over a 30-minute period on the mood VAS. Studies that have attempted to establish validity for the mood VAS have typically compared it with other measures of depression or negative affect. For example, studies that have examined individuals with major depression have largely found the mood VAS to correlate moderately to highly with the HRSD (Hamilton,

1960; as cited in Ahearn) with an average correlation coefficient of .62 across studies (reviewed in Ahearn).

6. *Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version* (SCID; First, Spitzer, Gibbon, & Williams, 1996). It is used to provide DSM-IV diagnoses. The major depression, mania, and dysthymia sections of the SCID will be completed for the proposed study. The SCID uses standardized clinician-directed queries for relevant symptomatic domains and has shown a high level of interrater reliability so that it has become the "gold standard" for diagnostic classification in clinical research settings. Segal et al. (1995) investigated the reliability of the SCID-I for providing a DSM-IV diagnosis for a MDE and found the Kappa, which corrects for chance agreement, to be .90, reflecting good agreement.

7. *State-Trait Anxiety Inventory* (STAI; Spielberger, 1968). It consists of 2 subscales, which assess state and trait anxiety independently. The trait scale of the STAI has demonstrated a test-retest reliability correlation coefficient that ranges from .73 to .86. The state anxiety scale has low test-retest reliability over time, which is expected given that it taps a transient state of anxiety. Moreover, it has been found that the state anxiety scale increases prior to surgery whereas trait anxiety remains relatively stable. This finding is consistent with predictions about the nature of state and trait anxiety where state anxiety is expected to increase in response to physical threat whereas trait anxiety would be unchanging (Finney, 1985). Only the state anxiety subscale was used in the present study.

Divided Visual Field Task Apparatus and Stimuli

All participants performed a divided visual field (DVF) task used in previous studies to examine dysfunctional cognitions in depression (e.g., Atchley et al., 2003; Atchley, Stringer, Mathias, Ilardi & Minatrea, in press). Stimuli for the DVF task were presented via an IBM compatible, Pentium-class computer with a Dell monitor and the E-prime program. Participants viewed the computer screen from a chin-rest, which helped minimize head movement and maintained vigilance to the fixation cross during the DVF task (e.g., so that words to the left of the fixation cross remain in the left visual field).

For this task, participants viewed prime and target word-pairs that were positive or negative adjectives (e.g., brave, loser). The prime word was presented in the center of the screen and was followed by a target word that was presented to either the left or right visual field. Participants were asked to make a valence judgment for the target word. The adjectives that made up the prime and target word-pairs were balanced for word length and production frequency (Kucera & Francis, 1967) and varied according to valence (positive vs. negative) based on previous norming research (Affective Norms for English Words: Bradley & Lang, 1999). Level of arousal produced by the words was controlled for by only using words with a moderate level of arousal (eliminating extremes) based on previous norming research (Bradley & Lang). On some trials, the prime and target word-pair were related and matched in terms of valence (e.g., brave followed by strong), whereas on other trials the prime and target word-pair were unrelated and did not match (e.g., brave followed by loser). The rationale for using a prime word followed by a target

word was to improve sensitivity of the task for detecting differences in accuracy and reaction time within a hemisphere. This is important because, regardless of mood, the left hemisphere will tend to be superior at this task because participants are being asked to read words (a verbal task for which the left hemisphere is superior; Banich, 1997). By using a prime, however, a comparison between related an unrelated trials can be made within a hemisphere.

Following a practice session consisting of 30 prime-target pairs, each participant was presented with 4 blocks of trials for the DVF task. Additionally, each block began with 4 practice trials (i.e., practice word pairs), and was followed by 96 trials that comprised the experiment. The first 2 experimental blocks consisted of words that were identical but were presented to a different visual field (e.g., "enraged" was presented to the left visual field in block 1 and the right visual field in block 2). A single trial for the DVF task consisted of a fixation cross, followed by a centrally presented prime word, followed by a flash mask (a series of number signs as an object mask). The flash mask was followed by a target word that was presented to only one visual field (left or right) and followed by another flash mask. Given that the participants performed the DVF task twice (in 2 blocks), once prior to mood induction or sham induction procedures and once following the induction procedures, two separate word lists (word list 1 and 2) were used. The adjectives that made up the two word lists (consisting of prime and targets adjectives) were matched for word length, production frequency, and valence (Kucera & Francis, 1967) based on previous norming research (Affective Norms for English Words: Bradley & Lang,

1999). Moreover, level of arousal produced by the words was controlled for by only using words with a moderate level of arousal based on previous norming research (Bradley & Lang). Finally, the two words lists were counterbalanced (to minimize order effects) so that some participants received word list 1 first, whereas other participants received word list 2 first.

Procedure

As discussed earlier, participants were recruited based on pretest responses to questionnaires administered through a mass prescreening measure on-line. At the study, participants completed a brief screening measure (IDD-L) to provide a probable diagnosis of depressed, previously depressed or never-depressed. Participants given a probable diagnosis of never-depressed were randomly assigned to one of two groups (one group underwent sad mood priming, the other group experienced a sham induction). Following the brief screening measure, participants performed the DVF task. Instructions were provided both visually (on the computer screen) and orally. Participants were instructed to attend to the fixation cross in the center of the screen and then read the prime word to themselves that followed the fixation cross. Moreover, the instructions informed participants that if they missed many of the targets, it is likely because they are shifting their gaze and they should, therefore, maintain focus on the fixation cross (presented central visual field). For the target words (presented to only one visual field), they were asked to make a valence judgment (i.e., determine whether the target is positive or negative) as quickly and accurately as possible by pressing a key on the computer with their right hand

(valence of words was determined with previous norming research, Bradley & Lang, 1999). Following the participant's response, feedback was provided (i.e., they were informed as to whether their response was correct, incorrect, or whether no response was detected). Participants were provided feedback to help them improve their accuracy on the task. By informing them when they are missing many targets, they are implicitly reminded to focus on the fixation cross rather than shift their gaze from one visual field to the other (suggested by the instructions). Completion of the *practice* block and first experimental block of the DVF task was followed with a brief assessment of mood using the VAS before participants began Block 2 for the task (same wordlist, but words were flipped in terms of visual field presentation).

Once the first two blocks of the DVF task (same list presented twice, but visual field presentation for targets was counterbalanced) was completed, previously depressed participants and one of the never-depressed groups of participants (i.e., the mood induction never-depressed participant group) underwent a sad mood induction procedure that has been used extensively in previous studies and has elicited a comparable sad mood in both groups (e.g., Ingram et al., 1994). The mood induction procedure combined a music induction and an autobiographical induction. The music induction used sad and nostalgic music played from the soundtrack for A Field of Dreams and the music was played continuously for 8 minutes. While participants listened to the music they were instructed to think about the saddest event in their lives. Currently depressed individuals did not undergo the mood induction procedure for ethical reasons. The other group of never-depressed participants also did not

undergo the mood induction procedure. These participant groups (depressed and the second never-depressed group) underwent a sham induction instead, which required that they listen to recorded tones and count these tones for the duration of 8 minutes. Following the induction procedures, all participants were administered the MAACL and VAS. These measures were used to confirm the sad-mood inducing effects of the mood induction procedure and to ensure that both groups (previously depressed and never-depressed mood induction groups) achieved the sad mood equally.

All participants performed the DVF task a second time (using the word list that was not used during the first DVF task administration) following the induction procedures. Following the first experimental block of the DVF task, the VAS was administered to ensure that the effects of the mood induction procedure remained. Finally, at the conclusion of the DVF task (completion of fourth block), the MAACL and VAS were re-administered to ensure that the sad mood was maintained equally by both groups. Depressed participants and never-depressed participants who do not undergo mood priming were administered these measures at each time point to maintain continuity between all participant groups.

Following the experimental portion of the study, participants completed the BDI-II to assess current depressive symptoms, and the STAI (Spielberger) to assess the level of anxiety. Following completion of these measures, an advanced graduate student in clinical psychology conducted the clinical interview (SCID) to confirm their depression status.

Design

The design for the primary experiment was a 2 (diagnostic group: previously depressed and mood-primed never-depressed participants) x 2 (visual field: right and left VF) x 2 (target valence: positive or negative) x 2 (prime-target relatedness; either related or unrelated prime-target pairings) x 2 (time: pre vs. post-mood induction) mixed analysis of covariance (ANCOVA) with state anxiety as a covariate. All variables were within-subjects factors with the exception of diagnostic group, which served as a between-subjects factor. Dependent variables were response accuracy and response time.

Results

Data Reduction

Overall accuracy across participant groups was relatively low, averaging 65% across previously depressed and mood-induced never depressed participants, and many participants systematically demonstrated poor accuracy – at or below chance – for specific study conditions (e.g., LVF/RH unrelated trials). Furthermore, because reaction-time data are typically analyzed for accurate trials only, and many participants demonstrated poor accuracy for specific study conditions, any reaction time findings associated with these conditions would be unreliable. Thus, reaction time data were not analyzed, and the primary analyses for the study focused on accuracy as the dependent variable.

Participant Characteristics

See Table 1 for descriptive statistics and means and standard deviations for the BDI and STAI-State measures for each diagnostic group included in the study analyses. Participants were all college students and predominantly in their late teen-age years. Moreover, formerly depressed participants were predominantly female, whereas participants in the never-depressed groups reflected greater numbers of male participants. Previously depressed and mood-primed never depressed participants were also compared for potential between-group differences on the BDI and STAI-State measures. Although there were no significant differences between the two aforementioned diagnostic groups in terms of BDI scores ($p = .072$), previously depressed participants reported experiencing greater state-anxiety (STAI-State

average total score was 37) relative to mood-primed never depressed participants (STAI-State average total score was 31), which is supported by their significantly higher STAI-state scores, $t(46) = 2.284$, $p = .027$. It is also worth noting that although none of the participants were currently depressed, approximately one-third reported significant state-anxiety, which is reflected in their sufficiently elevated scores on the STAI-state measure (31.25% of previously depressed and mood-induced never depressed participants had scores of 40 or higher on the STAI-State). Table 2 provides correlations among all of the self-report depressive and anxious affect measures used in the present study, which largely correlated with one another.

Table 1.

Participant Descriptives

Variable	Formerly Depressed (n = 27)	Mood-Induced Never Depressed (n = 21)	Sham-Induced Never Depressed (n = 19)
Age	19.72 (1.60)	18.95 (.97)	19.32 (.95)
Gender	9M/18F	11M/10F	13M/6F
BDI-II	5.52 (3.01)	3.90 (3.03)	2.84 (3.15)
STAI-S	36.74 (8.84)	31.05 (8.20)	28 (8.25)

Note. Means and (standard deviations) for age, and self-reported depression and anxiety questionnaires. M = male; F = female. BDI-II = Beck Depression Inventory (Beck, Steer & Brown, 1996); STAI-S = State-Trait Anxiety Inventory, State Anxiety Subscale (Spielberger, 1968).

Table 2.

Intercorrelations Among Depressive Symptom Severity, State Depression and State Anxiety Self-Report Measures.

Measure	BDI-II	STAI-S	MAACL-R-D	VAS
		Participants (N = 67)		
BDI-II	__	.42*	.22	.27*
STAI-S	.42*	__	.41*	.43*
MAACL-R-D	.22	.41*	__	.60*
VAS	.27*	.43*	.60*	__

Note: BDI-II = Beck Depression Inventory (Beck, Steer & Brown, 1996); STAI-S = State-Trait Anxiety Inventory, State Anxiety Subscale (Spielberger, 1968), MAACL-R-D = Multiple Affect Adjective Checklist-Revised-Depression Subscale (Zuckerman & Lubin, 1965), VAS = Visual Analogue Scale (Grossberg & Grant, 1978).

* $p < .05$.

Manipulation Check

To confirm that the mood induction procedure was comparably successful at inducing a sad mood state for the previously depressed and never-depressed participants, and that the induced sad mood was maintained throughout the experiment, the VAS and MAACL measures were examined across time for both participant groups. Moreover, because a pre-mood induction measure of the MAACL (i.e., baseline measure) was not included in this study, never-depressed participants

who experienced the sham-induction were also included in the omnibus analysis for this measure (as a between-groups comparison) to evaluate the effectiveness of the mood-priming procedure in inducing a temporary sad mood state. For the MAACL, a 3 (Group: previously depressed, mood induction never-depressed, and sham-induction never depressed groups) x 2 (Time: immediately after induction procedures, end of the second DVF task administration) mixed analysis of variance (ANOVA) was conducted. Likewise, a 2 (Group: previously depressed and never-depressed mood-induced groups) x 4 (Time) mixed ANOVA was conducted for the VAS. (Time 1 was pre-induction, following the first DVF block; Time 2 refers to the administration after mood induction and following second DVF block; Time 3 was after the third experimental block of the DVF task; and Time 4 refers to the period following administration of the fourth DVF block). In both analyses, Group was the between-subjects factor and Time was the within-subjects factor. It was predicted that previously depressed and never-depressed participants who underwent the mood-priming procedure would demonstrate significantly higher scores on the depression subscale of the MAACL at both time points relative to the sham-induced never-depressed participants. Moreover, it was predicted that there would be no significant differences on the MAACL between previously depressed and mood-primed never-depressed participants, and no change over time for any of the groups. For the VAS, it was predicted that previously depressed and mood-primed never-depressed participants would demonstrate significantly greater negative affect at Time 2 (after mood priming), Time 3 (after the third experimental block on the DVF task), and

Time 4 (following the fourth and final block of DVF task) relative to their VAS scores at Time 1 (pre-mood induction), with no significant differences between the two groups, and no significant differences between Time 2, Time 3, and Time 4.

The omnibus ANOVA for the MAACL revealed a significant main effect for Group, $F(2, 64) = 20.241$, $p < .001$, and a significant Time-by-Group interaction, $F(2, 64) = 14.848$, $p < .001$. Consistent with study hypotheses, follow-up planned comparisons showed that previously depressed participants demonstrated a significantly higher score on the depression subscale of the MAACL compared with never-depressed participants who underwent the sham induction at Time 1, $F(1, 64) = 65.221$, $p < .001$, and Time 2, $F(1, 64) = 11.875$, $p = .001$. Furthermore, mood-primed never-depressed participants also demonstrated significantly higher depression subscale scores relative to sham-induced never-depressed participants at Time 1, $F(1,64) = 26.423$, $p < .001$, and Time 2, $F(1, 64) = 9.149$, $p = .004$. It was also found, however, that previously depressed participants scored significantly higher on the depression subscale relative to mood-primed never depressed participants at Time 1, $F(1, 64) = 7.387$, $p = .008$, which is inconsistent with study predictions (i.e., that previously depressed and mood primed never depressed groups would demonstrate comparably sad mood at both time points), but these group differences were no longer significant at Time 2 ($p = .8$). Lastly, all follow-up comparisons remained significant even after the alpha level was adjusted for multiple comparisons (Dunn Sidak adjusted $\alpha = .017$ for $c = 3$ comparisons). Results of this

analysis are presented in Table 3, and means and standard deviations for the MAACL are provided in Table 4.

Table 3

Analysis of Variance for Depression Subscale of the MAACL-R

Source	*df*	*F*	*p*
	Between Subjects		
Group	2	20.241**	.01
S within-group Error	64	(33.408)	
	Within Subjects		
Time	1	3.861	.05
Time x Group	2	14.848**	.01
Error	64	(6.63)	

Note: Multiple Affect Adjective Checklist-Revised = MAACL-R (Zuckerman & Lubin, 1965). Values enclosed in parentheses represent mean square errors. S = subjects.

* $p < .05$. ** $p < .01$.

Table 4.

Mean Total Scores and Standard Deviations for Depressive Affect After Induction as a Function of Time and Group

	Time 1			Time 2		
MAACL-R	M	SD	N	M	SD	n
Formerly Depressed	21.70_{a}	3.55	27	17.81_{a}	3.52	27
Mood Primed Never Depressed	18.24_{b}	4.75	21	17.48_{a}	4.88	21
Sham Induced Never Depressed	11.11_{c}	4.50	19	13.11_{b}	5.46	19

Note. Depressive affect scores are from the depression subscale of the Multiple Affect Adjective Checklist-Revised (MAACL-R; Zuckerman & Lubin, 1965). Time 1 = immediately following induction procedures, Time 2 = end of divided visual field task. Means in the same column that do not share subscripts differ at $p < .05$ in the Dunn Sidak significant difference comparison.

Because depressive affect was of primary interest in the present study, an additional analysis for the MAACL depression subscale was conducted to examine whether depressive affect – that is relatively independent of general negative affect (e.g., anxiety) – was uniquely affected by the mood induction. Toe examine the effect of the induction on depressive affect specifically, variance associated with the anxiety and hostility subscale scores was removed from the depression subscale using a multiple regression model to generate residualized depression scores (i.e., the variance remaining in the depression subscale after the variance associated with anxiety and hostility was removed), whereby the depression subscale was the

criterion variable and the hostility and anxiety subscales were the predictor variables. Results of these regression analyses are presented in Table 5. These residualized depression scores, in turn, were used as the dependent variable (in lieu of the full depression subscale) in the repeated measures ANOVA model that was previously described for the MAACL analysis; the 3 (Group) x 2 (Time) repeated measures ANOVA. This omnibus ANOVA revealed a significant main effect for Group, $F(2, 64) = 16.56$, $p < .001$, and a significant Time-by-Group interaction, $F(2, 64) = 6.029$, $p = .004$. Follow-up planned comparisons for this analysis revealed that previously depressed participants continued to demonstrate significantly higher scores on the residualized depression subscale of the MAACL compared with sham-induced never depressed participants at Time 1, $F(1, 64) = 39.181$, $p < .001$, and Time 2, $F(1, 64) = 13.723$, $p < .001$. Furthermore, mood-primed never depressed participants also demonstrated significantly higher residualized depression subscale scores relative to sham-induced never depressed participants at Time 1, $F(1, 64) = 20.964$, $p < .001$, and Time 2, $F(1, 64) = 10.943$, $p = .002$. Notably, there were no significant differences between the previously depressed and mood-primed never depressed participants on the residualized depression subscale scores at either time point. Furthermore, all follow-up comparisons remained significant even after the alpha level was adjusted for multiple comparisons (Dunn Sidak adjusted $\alpha = .017$ for $c = 3$ comparisons). Results of this analysis are presented in Table 6.

Table 5.

Summary of Regression Analysis for Contribution of Anxiety and Hostility Subscales to Depression Subscale Scores for the MAACL (N = 67)

Variable	*B*	*SE B*
Time 1		
Anxiety	1.74	.81
Hostility	.63	.71
Time 2		
Anxiety	1.30	.99
Hostility	.63	.62

Note: Anxiety, hostility, and depression affect scores are from the depression subscale of the Multiple Affect Adjective Checklist-Revised (Zuckerman & Lubin, 1965). Time 1 = immediately following induction procedures, and Time 2 = end of divided visual field experiment.

Table 6.

Analysis of Variance for Residual Depression Subscale Scores for the MAACL

Source	*df*	*F*	*p*
	Between Subjects		
Group	2	16.56**	.01
S within-group error	64	(33.47)	
	Within Subjects		
Time	1	.24	.63
Time x Group	2	6.03**	.01
Error	64	(7.21)	

Note: Depressive affect scores are from the depression subscale of the Multiple Affect Adjective Checklist-Revised (Zuckerman & Lubin, 1965). Values enclosed in parentheses represent mean square errors. S = subjects.

* $p < .05$. ** $p < .01$.

Because the omnibus ANOVA model for the VAS did not meet the sphericity assumption, the Huynh-Feldt adjustment to the df was used. A significant main effect for Time was observed, $F(2.414, 111.061) = 27.082$, $p < .001$. Notably there was not a significant Time-by-Group interaction or a main effect for Group, suggesting that the mood induction produced a similar effect on previously depressed and never-depressed participants. Moreover, VAS scores at Time 2 and Time 3 showed

participants (in both groups) to be significantly sadder relative to their Time 1 scores (i.e., VAS scores were larger at Time 2 and Time 3 compared with Time 1 scores); $F(1, 46) = 49.1$, $p < .001$ and $F(1, 46) = 15.232$, $p < .001$, respectively. However, comparisons examining the VAS scores at Time 4 revealed a non-significant difference between Time 1 and Time 4 scores, $p = .057$, suggesting that the sad mood had dissipated by the end of the experiment. Furthermore, examination of VAS scores following mood priming suggested a linear decline in VAS scores, whereby the sad mood gradually dissipated over time because participants scores were lower (i.e., less sad) at Time 3 relative to Time 2, $F(1, 46) = 20.033$, $p < .001$, and Time 4 relative to Time 3, $F(1, 46) = 20.856$, $p < .001$. Additionally, all follow-up comparisons remained significant even after the alpha level was adjusted for multiple comparisons (Dunn Sidak adjusted $\alpha = .008512$ for $c = 6$ comparisons). Thus, although the VAS scores supported significant sad mood priming with the induction procedure that produced a similar effect for both participant groups, later VAS scores suggested that the sad mood wore off and participants' reported mood state was no longer significantly different from baseline (i.e., prior to the mood priming procedure). Results of this analysis are presented in Table 7, and means and standard deviations for the VAS are provided in Table 8.

Table 7

Analysis of Variance for the VAS

Source	*df*	*F*	*p*
	Between Subjects		
Group	1	.13	.73
S within-group error	46	(11.52)	
	Within Subjects		
Time	2.41	27.08**	.01
Time x Group	2.41	2.26	.10
Error	111.06	(7.21)	

Note: VAS = Visual Analogue Scale (Grossberg & Grant, 1978). Values enclosed in parentheses represent mean square errors. S = subjects. A Huynh-Feldt adjustment was used for the *df* because the model did not meet the sphericity assumption for the analysis of variance test.

* $p < .05$. ** $p < .01$.

Table 8.

Mean Total Scores and Standard Deviations for Sad Mood as a Function of Time and Diagnostic Group.

	Formerly Depressed			Mood-Primed Never Depressed		
VAS	M	SD	N	M	SD	n
			Before Mood Induction			
Time 1	1.83 $_{a}$	1.91	27	1.99 $_{a}$	1.92	21
			After Mood Induction			
Time 2	5.00$_{b}$	2.63	27	3.99 $_{b}$	2.08	21
Time 3	3.35 $_{b}$	2.27	27	3.03 $_{b}$	1.92	21
Time 4	2.24 $_{a}$	1.99	27	2.71 $_{a}$	2.05	21

Note. Sad mood scores are based on subjects' Visual Analogue Scale ratings (VAS; Grossberg & Grant, 1978). Time 1 = prior to induction procedures, Time 2 = immediately following induction procedures, Time 3 = following the third block of the divided visual field task, and Time 4 = end of divided visual field experiment. Means in the same column that do not share subscripts with Time 1 VAS scores differ at $p < .05$ in the Dunn Sidak significant difference comparison.

In summary, the VAS and MAACL measures largely provided support for the efficacy of the mood induction procedure in producing a comparably sad mood state for previously depressed and never-depressed participants (i.e., the VAS and MAACL scores supported an immediate effect of sad mood priming for both groups

of participants). According to the MAACL, however, mood priming may have initially produced greater global negative affect (e.g., greater anxiety and/or hostility) in previously depressed participants, but it was associated with comparable depressive affect across the diagnostic groups. Moreover, this between-group difference wore off relatively quickly (i.e., previously depressed participants did not score significantly higher than mood primed never-depressed participants on the full depression subscale for the second MAACL administered), and VAS scores suggested that the procedure produced a similar effect for both groups of participants. Furthermore, VAS scores suggested that the sad mood state wore off relatively quickly - given the brief duration of the experiment – because final VAS scores were not significantly different from baseline (pre-induction) for either group. Depression (and residualized depression) subscale scores from the second MAACL administered, however, suggested that there may have been an enduring effect of the mood induction procedure because both groups (previously depressed and mood primed never depressed participants) continued to demonstrate greater subscale (and residualized subscale) scores relative to sham-induced never-depressed participants. Given the mixed results from the VAS and MAACL scores – particularly the dissipating sad mood state that was associated with the VAS and not the MAACL - it is highly probable that these measures are tapping different aspects associated with a sad mood state.

Tests for Fatigue, Practice, and Wordlist Effects

In order to examine the effects of mood priming on DVF task performance, it was also important to ensure that the two word lists were equivalent and that there were no significant fatigue or practice effects for the task over time so that any observed differences in performance following mood priming could be attributed to a sad mood and not one of the aforementioned effects. Thus, two separate repeated measures ANOVA models were conducted to test for these effects with sham induced never-depressed participants for the primary dependent variable of interest – accuracy. First, a 2 (Time: first administration of the DVF task for blocks 1 and 2, second administration of the DVF task for blocks 3 and 4) x 2 (VF: right and left) x 2 (target Valence: positive or negative) x 2 (Relatedness: related prime-target pair or unrelated) repeated measures ANOVA was conducted with sham-induced, never-depressed participants to test for practice and fatigue effects. It was expected that there would be no effect of time on accuracy rates (i.e., no main effects or interactions with Time), which would be consistent with no practice or fatigue effects. Results of this analysis are presented in Table 9, and means and standard deviations are provided in Table 10. The analysis revealed no significant effect of Time (main effect, $p = .475$) on DVF task accuracy rates, and Time did not significantly interact with any of the other independent variables.

Table 9

Analysis of Variance for Fatigue and Practice Effects

Source	*df*	*F*	*p*
	Within Subjects		
Time	1	.53	.48
S within-group error	18	(.02)	
VF	1	28.70**	.01
S within-group error	18	(.03)	
Valence	1	.75	.40
S within-group error	18	(.03)	
Relatedness	1	4.47*	.05
S within-group error	18	(.02)	
Time X VF	1	.37	.55
S within-group error	18	(.01)	
Time X Valence	1	.25	.63
S within-group error	18	(.01)	
VF X Valence	1	2.35	.14
S within-group error	18	(.13)	
Time X VF X Valence	1	1.23	.28
S within-group error	18	(.01)	

Table 9 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
Time X Related	1	.10	.76
S within-group error	18	(.01)	
VF X Related	1	.05	.82
S within-group error	18	(.01)	
Time X VF X Related	1	3.98	.06
S within-group error	18	(.01)	
Valence X Related	1	5.27*	.03
S within-group error	18	(.01)	
Time X Valence X Related	1	1.38	.26
S within-group error	18	(.01)	
VF X Valence X Related	1	.01	.97
S within-group error	18	(.01)	
Time X VF X Valence X Related	1	1.54	.23
S within-group error	18	(.01)	

Note: Values enclosed in parentheses represent mean square errors. S = subjects.

* $p < .05$. ** $p < .01$.

Table 10.

Mean Accuracy Rates and Standard Deviations as a Function of Time, Visual Field, Valence, and Prime-Target Relationship.

	LVF				RVF			
	Positive		Negative		Positive		Negative	
Relatedness	M	SD	M	SD	M	SD	M	SD
				Before Induction				
Related	61	.22	70	.17	76	.14	72	.19
Unrelated	58	.23	62	.18	79	.16	66	.24
				After Induction				
Related	61	.22	66	.16	81	.13	74	.15
Unrelated	63	.22	63	.18	70	.12	68	.17

Note. Sham-induced control subjects, n = 19. LVF = left visual field, RVF = right visual field. Accuracy rates reflect percent of correctly identified targets.

The second model tested for wordlist effects and was a 2 (Wordlist: wordlist 1 or wordlist 2) x 2 VF (right and left VF) x 2 (target valence: positive or negative) x 2 (relatedness: related prime-target pair or unrelated) repeated measures ANOVA for the accuracy dependent variable. It was expected that there would be no effect of wordlist on task performance for accuracy rates (i.e., no main effect and no interactions), which would be consistent with wordlist equivalency. Results of this analysis are presented in Table 11, and means and standard deviations are provided in Table 12. The analysis revealed no significant differences between the wordlists on

overall accuracy rates ($p = .116$). However, wordlist significantly interacted with the prime-target relatedness independent variable, $F(1, 18) = 6.861$, $p = .017$. Examination of this 2-way interaction revealed that sham induced never-depressed participants demonstrated higher accuracy on prime-target related trials for wordlist 1 (72.4%) compared with wordlist 2 (67.6%). However, this difference between the wordlists didn't affect overall accuracy rates, which is reflected by the non-significant main effect for Wordlist. Nonetheless, if the primary analyses for the study examining accuracy rates for previously depressed and mood primed never depressed participants revealed a significant effect for the Time independent variable – particularly if it interacted with Relatedness - findings would need to be interpreted with caution and statistically controlled to permit interpretation of mood priming effects.

Table 11

Analysis of Variance for Wordlist Effects

Source	*df*	*F*	*p*
	Within Subjects		
Wordlist	1	2.72	.12
S within-group error	18	(.02)	
VF	1	28.70**	.01
S within-group error	18	(.03)	
Valence	1	.75	.40
S within-group error	18	(.03)	
Relatedness	1	4.47*	.05
S within-group error	18	(.02)	
Wordlist X VF	1	2.71	.117
S within-group error	18	(.01)	
Wordlist X Valence	1	.33	.58
S within-group error	18	(.01)	
VF X Valence	1	2.35	.14
S within-group error	18	(.13)	
Wordlist X VF X Valence	1	2.81	.11
S within-group error	18	(.01)	

Table 11 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
Wordlist X Related	1	6.86*	.02
S within-group error	18	(.01)	
VF X Related	1	.05	.82
S within-group error	18	(.01)	
Wordlist X VF X Related	1	1.96	.18
S within-group error	18	(.01)	
Valence X Related	1	5.27*	.03
S within-group error	18	(.01)	
Wordlist X Valence X Related	1	2.95	.10
S within-group error	18	(.01)	
VF X Valence X Related	1	.01	.97
S within-group error	18	(.01)	
Wordlist X VF X Valence X Related	1	.05	.82
S within-group error	18	(.01)	

Note: Values enclosed in parentheses represent mean square errors. S = subjects.

* $p < .05$. ** $p < .01$.

Table 12.

Mean Accuracy Rates and Standard Deviations as a Function of Wordlist, Visual Field, Valence, and Prime-Target Relationship.

	LVF				RVF			
	Positive		Negative		Positive		Negative	
Relatedness	M	SD	M	SD	M	SD	M	SD
				Wordlist 1				
Related	62	.22	72	.17	81	.12	74	.16
Unrelated	62	.24	64	.15	78	.13	63	.22
				Wordlist 2				
Related	60	.21	64	.15	75	.15	71	.18
Unrelated	59	.21	61	.21	77	.15	70	.18

Note. Sham-induced control subjects, n = 19. LVF = left visual field, RVF = right visual field. Accuracy rates reflect percent of correctly identified targets.

Preliminary Analyses for Divided Visual Field Task

Because the anxiety covariate did not significantly interact with any of the other independent variables - and there was no main effect for this variable – anxiety was subsequently removed from the model and the omnibus analysis was re-run using a mixed ANOVA model. Results of this analysis are presented in Table 13. Thus, accuracy rates were analyzed in a 2 (Group: previously depressed and mood-primed never-depressed participants) x 2 (VF: right and left) x 2 (target valence: positive or

negative) x 2 (prime-target relatedness) x 2 (Time: pre vs. post-mood induction [Time 1 vs. Time 2) mixed analysis of variance (ANOVA). For all planned and post-hoc analyses, an alpha level of .05 was used as the critical value.

Table 13

Analysis of Variance for Accuracy Rates Among Previously Depressed and Mood-Primed Never Depressed Participants with Varied Degrees of State-Anxiety (N = 48)

Source	*df*	*F*	*p*
	Between Subjects		
Group	1	4.24	.05
STAI-S	1	.75	.39
S within-group error	45	(.10)	
	Within Subjects		
Time	1	.12	.74
Time X STAI-S	1	.75	.39
Time X Group	1	.02	.89
S within-group error	45	(.02)	
VF	1	19.83**	.01
VF X STAI-S	1	1.88	.18
VF X Group	1	.07	.80
S within-group error	45	(.03)	
Valence	1	.31	.58

Table 13 (continued).

Source	*df*	*F*	*P*
	Within Subjects		
Valence X STAI-S	1	.37	.55
Valence X Group	1	.62	.43
S within-group error	45	(.04)	
Relatedness	1	1.95	.17
Relatedness X STAI-S	1	.08	.77
Relatedness X Group	1	.19	.67
S within-group error	45	(.03)	
Time X VF	1	.64	.43
Time X VF X STAI-S	1	.37	.55
Time X VF X Group	1	.05	.82
S within-group error	45	(.01)	
Time X Valence	1	1.15	.29
Time X Valence X STAI-S	1	.93	.34
Time X Valence X Group	1	.36	.55
S within-group error	45	(.01)	
VF X Valence	1	.07	.80
VF X Valence X STAI-S	1	.01	.96
VF X Valence X Group	1	.07	.79

Table 13 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
S within-group error	45	(.03)	
Time X VF X Valence	1	.07	.80
Time X VF X Valence X STAI- S	1	.02	.89
Time X VF X Valence X Group	1	.62	.44
S within-group error	45	(.02)	
Time X Related	1	.70	.41
Time X Related X STAI-S	1	.78	.38
Time X Related X Group	1	.90	.35
S within-group error	45	(.02)	
VF X Related	1	1.39	.25
VF X Related X STAI-S	1	.83	.37
VF X Related X Group	1	2.99	.09
S within-group error	45	(.01)	
Time X VF X Related	1	.22	.64
Time X VF X Related X STAI-S	1	.37	.55
Time X VF X Related X Group	1	.24	.63

Table 13 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
S within-group error	45	(.01)	
Valence X Related	1	.40	.53
Valence X Related X STAI-S	1	.01	.93
Valence X Related X Group	1	.76	.39
S within-group error	45	(.01)	
Time X Valence X Related	1	.04	.85
Time X Valence X Related X STAI-S	1	.02	.90
Time X Valence X Related X Group	1	.03	.87
S within-group error	45	(.01)	
VF X Valence X Related	1	3.18	.08
VF X Valence X Related X STAI-S	1	1.72	.20
VF X Valence X Related X Group	1	3.71	.06
S within-group error	45	(.01)	
Time X VF X Valence X Related	1	.97	.33
Time X VF X Valence X Related X STAI-S	1	1.41	.24

Table 13 (continued).

Source	*df*	*F*	*p*
Time X VF X Valence X Related X Group	1	.07	.79
S within-group error	45	(.01)	

Note: Values enclosed in parentheses represent mean square errors. S = subjects. State-Trait Anxiety Inventory, State Anxiety Subscale (Spielberger, 1968).

* $p < .05$. ** $p < .01$.

The omnibus ANOVA revealed a significant main effect for visual field (VF) for the target presentation, $F(1, 46) = 163.247$, $p < .001$, indicating that words presented to the RVF/LH were more accurately judged (73.19%) than words presented to the LVF/RH (57.65%). Moreover, there were several additional main effects and interaction terms that were found to be statistically significant, however, given the very large magnitude of the VF main effect, these findings are extremely suspect. In fact, VF main effects associated with the DVF task are typically on the order of a 10% difference in accuracy (e.g., Banich, 1997), and the present study evidenced a VF main effect difference on the order of 16%, which suggests something unusual about the task performance for many of the participants in the present study. There are a number of possible explanations for this abnormally large VF main effect, which will be reviewed in the discussion, however, it seems clear that many participants in the present study performed very poorly on LVF/RH trials relative to RVF/LH trials as is evidenced by their very poor LVF/RH accuracy rate (57.65%) compared with their predictable RVF/LH accuracy rate (73.19%); Atchley

and colleagues (2005) observed a LVF/RH accuracy rate of 65%, and RVF/LH accuracy rate of 74%. Therefore, in order to address the very poor LVF/RH accuracy rate (which was not much greater than chance across participants) – and indirectly address the unusually large VF main effect - participants who did not perform significantly better than chance for LVF/RH trials were excluded from all subsequent analyses, and the omnibus ANOVA was re-run without these participants.

Divided Visual Field Task Analyses - Accuracy

Exclusion of participants with poor accuracy for LVF/RH trials eliminated many participants from the analysis, and therefore, made it challenging to identify statistically significant findings because of very low power; 10 previously depressed and 13 mood-induced never-depressed participants remained in the model. However, there were still a limited number of statistically significant findings when the mixed ANOVA model was re-run using only participants with accuracy rates that were significantly better than chance for the LVF/RH trials.

This revised omnibus ANOVA model (with 23 participants) revealed a significant main effect for VF, $F(1, 21) = 65.579$, $p < .001$, indicating that words presented to the RVF/LH were more accurately judged (78%) than words presented to the LVF/RH (65%). Moreover, there was a significant main effect for Relatedness (prime-target relationship), $F(1, 21) = 9.977$, $p = .005$, such that participants responded with greater accuracy to related trials (74%) than unrelated trials (69%). The Relatedness main effect, however, is best subsumed by the significant two-way interaction between Valence and Relatedness, $F(1, 21) = 4.991$, $p = .036$. Results of

this analysis are presented in Table 14, and means and standard deviations are provided in Table 15.

Table 14

Analysis of Variance for Accuracy Rates Among Previously Depressed and Mood-Primed Never Depressed Participants (N = 23)

Source	*df*	*F*	*p*
	Between Subjects		
Group	1	1.64	.22
S within-group error	21	(.08)	
	Within Subjects		
Time	1	2.07	.17
Time X Group	1	.36	.56
S within-group error	21	(.02)	
VF	1	65.58**	.01
VF X Group	1	1.95	.18
S within-group error	21	(.02)	
Valence	1	.18	.68
Valence X Group	1	.53	.48
S within-group error	21	(.03)	
Relatedness	1	9.98**	.01
Relatedness X Group	1	.03	.87

Table 14 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
S within-group error	21	(.03)	
Time X VF	1	.23	.64
Time X VF X Group	1	.01	.98
S within-group error	21	(.01)	
Time X Valence	1	.06	.81
Time X Valence X Group	1	.01	.98
S within-group error	21	(.01)	
VF X Valence	1	2.37	.14
VF X Valence X Group	1	.07	.80
S within-group error	21	(.02)	
Time X VF X Valence	1	.35	.56
Time X VF X Valence X Group	1	.39	.54
S within-group error	21	(.01)	
Time X Related	1	.11	.75
Time X Related X Group	1	1.40	.25
S within-group error	21	(.01)	
VF X Related	1	2.98	.10
VF X Related X Group	1	3.49	.08

Table 14 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
S within-group error	21	(.01)	
Time X VF X Related	1	.33	.57
Time X VF X Related X Group	1	.08	.78
S within-group error	21	(.01)	
Valence X Related	1	4.99*	.04
Valence X Related X Group	1	.13	.72
S within-group error	21	(.01)	
Time X Valence X Related	1	.14	.71
Time X Valence X Related X Group	1	.16	.70
S within-group error	21	(.01)	
VF X Valence X Related	1	2.85	.11
VF X Valence X Related X Group	1	.47	.50
S within-group error	21	(.01)	
Time X VF X Valence X Related	1	4.01	.06
Time X VF X Valence X Related X Group	1	.09	.77
S within-group error	21	(.01)	

Note: Values enclosed in parentheses represent mean square errors. S = subjects.

* $p < .05$. ** $p < .01$.

Table 15

Mean Accuracy Rates and Standard Deviations as a Function of Time, Visual Field, Valence, Prime-Target Relationship, and Group.

	Previously Depressed				Never Depressed			
	Positive		Negative		Positive		Negative	
	LVF	RVF	LVF	RVF	LVF	RVF	LVF	RVF
	Before Induction							
Related								
M	64	76	71	74	60	84	74	.16
SD	.11	.14	.14	.13	.16	.15	63	.22
Unrelated								
M	60	72	56	70	64	77	63	77
SD	.12	.11	.12	.15	.15	.11	.11	.11
	After Induction							
Related								
M	67	76	70	77	68	84	71	85
SD	.13	.15	.12	.16	.15	.08	.13	.06
Unrelated								
M	63	80	62	74	61	79	63	78
SD	.13	.11	.13	.17	.15	.12	.20	.12

Note. Previously depressed subjects, n = 10, never-depressed subjects, n = 13. LVF = left visual field, RVF = right visual field. Accuracy rates reflect percent of correctly identified targets.

Examination of this two-way interaction revealed a related-trial advantage for negative targets, $t(22) = 4.121$, $p < .001$, such that negative targets that were preceded by a related prime word (i.e., a negative prime word) were judged more accurately (76%) than negative targets preceded by an unrelated prime word (68%). The pattern of results for positive targets, however, trended in the predicted direction (i.e., positive target words preceded by a related prime word were judged more accurately than positive target words preceded by an unrelated prime), but was not statistically significant, $p = .118$.

Accuracy Rate and Length of Time Since the Most Recent Major Depressive Episode

In addition to the primary analysis for accuracy, which compared diagnostic groups, a repeated measures ANCOVA was conducted for previously depressed participants - with above chance accuracy rates for the LVF/RH - to determine whether the length of time that had lapsed since their most recent major depressive episode (MDE) influenced their performance on the DVF task. The design for this analysis was a 2 (VF: right and left) x 2 (Valence: positive or negative) x 2 (Relatedness: related or unrelated) x 2 (Time: pre vs. post-mood induction) ANCOVA with the number of months since their most recent MDE episode included as a covariate. All variables were within subjects factors, and accuracy was the dependent variable. This analysis revealed no significant interactions for any of the

independent variables, and there was no evidence to support a significant influence of length of time since the most recent MDE on participants' valence judgments for negative and positive words. However, given the very small sample size (n = 10) for this analysis, the lack of significant findings was not unexpected.

Table 16

Analysis of Variance for Accuracy Rates Among Previously Depressed Participants in Relation to Number of Months in Recovery from Depression (N = 10)

Source	*df*	*F*	*p*
	Between Subjects		
Months	1	1.42	.27
S within-group error	8	(.07)	
	Within Subjects		
Time	1	.07	.79
Time X Months	1	2.28	.17
S within-group error	8	(.02)	
VF	1	5.37	.05*
VF X Months	1	2.05	.19
S within-group error	8	(.03)	
Valence	1	.08	.78
Valence X Months	1	.07	.80
S within-group error	8	(.04)	

Table 16 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
Relatedness	1	5.14	.05
Relatedness X Months	1	.19	.67
S within-group error	8	(.02)	
Time X VF	1	.40	.55
Time X VF X Months	1	2.21	.18
S within-group error	8	(.01)	
Time X Valence	1	.75	.41
Time X Valence X Months	1	1.71	.23
S within-group error	8	(.01)	
VF X Valence	1	.07	.80
VF X Valence X Months	1	.77	.41
S within-group error	45	(.01)	
Time X VF X Valence	1	.43	.53
Time X VF X Valence X Months	1	1.34	.28
S within-group error	8	(.02)	
Time X Related	1	.46	.52
Time X Related X Months	1	.14	.72
S within-group error	8	(.01)	

Table 16 (continued).

Source	*df*	*F*	*p*
	Within Subjects		
VF X Related	1	3.45	.10
VF X Related X Months	1	.54	.48
S within-group error	8	(.01)	
Time X VF X Related	1	.01	.92
Time X VF X Related X Months	1	.01	.96
S within-group error	8	(.01)	
Valence X Related	1	12.63	.01**
Valence X Related X Months	1	4.79	.06
S within-group error	8	(.01)	
Time X Valence X Related	1	.01	.99
Time X Valence X Related X Months	1	.01	.10
S within-group error	8	(.01)	

Note: Values enclosed in parentheses represent mean square errors. S = subjects.

Months = Number of months since the most recent major depressive episode.

* $p < .05$. ** $p < .01$.

Discussion

The primary aim of this study was to examine the interrelationships among mood state, depressive experience, and anxious symptomatology in the processing of emotional words using a divided visual field paradigm. Specifically, this study attempted: (a) to replicate the findings of Atchley et al. (2003, 2005), who observed results consistent with an enduring depressive verbal processing bias in the right hemisphere, even in the absence of sad mood; and (b) to provide answers to remaining questions about the potential influence of state dysphoria and state anxiety on emotional processing. This study replicated the well-established finding of a left hemispheric/RVF advantage for language processing – as evidenced by participants' higher accuracy rates for all words presented to the RVF. Moreover, the well-established processing advantage on a valence judgment task for valence-primed words was also supported, inasmuch as negative words that were preceded by a valence-related prime were judged more accurately. The present study, however, failed to replicate previously reported findings of superior right hemispheric (RH) accuracy judgments, and of a larger valence-priming advantage for negative words among previously depressed participants relative to never-depressed participants (Atchley et al., 2003 & 2005).

Although the present study failed to replicate Atchley and colleagues' previous findings (2003 & 2005), it is not surprising given that the majority of participants demonstrated exceptionally poor accuracy for all words presented to the LVF/RH, and therefore, were excluded from further analyses. Consequently, the

analyses for the present study possessed very limited power for detecting reliable differences in performance on the DVF task within individual participants and between diagnostic groups. Thus, the null findings associated with the present study should not be accepted as indicative of what would be found in studies with larger samples sizes and greater statistical power, and furthermore, should *not* be interpreted as an appropriate comparison to Atchley and colleagues' previous studies because of its limited power (2003 & 2005). However, given that the present study attempted to replicate Atchley and colleagues' previous findings – among several other stated study aims - it seems paramount to identify possible explanations for the present study's poor LVF/RH accuracy rate, which led to the exclusion of many participants and contributed to the study's null findings (because of limited statistical power).

One possible explanation for the poor RH accuracy rate relates to the nature of the DVF task, which is challenging largely because targets are presented for a very short duration to reduce the likelihood that participants move their eyes between trials (which could result in targets not being projected to the intended hemisphere). Because of the inherent difficulty associated with the DVF task, participants typically require some practice in order to gain proficiency at it. Although participants in the present study engaged in a practice session (consisting of 30 trials) and 4 practice trials at the beginning of each block, it is quite possible that they did not have sufficient practice with it, and therefore, their performance was compromised during the experiment; overall accuracy for the present study was 65% across diagnostic groups and VFs compared with an average of 73% for Atchley and colleagues'

previous studies combined. Notably, task difficulty and insufficient practice would tend to disproportionately affect participants' ability to accurately detect word targets for LVF/RH trials relative to RVF/LH trials because left hemisphere word targets are generally easier to identify (i.e., the left hemisphere is superior at language comprehension in 95% of right-handed individuals, see Rasmussen & Milner, 1977b). Thus, task difficulty combined with insufficient practice likely contributed to the poor accuracy rate observed for LVF/RH trials in the present study.

Additionally, it remains possible that the feedback provided with each trial – where participants were informed as to whether their response was correct, incorrect, or not detected –inadvertently contributed to some of the participants' poor accuracy rates because it was experienced as stressful (e.g., participants observed that they were incorrect at detecting targets on multiple trials). In fact, numerous studies have demonstrated a negative correlation between high levels of stress/arousal and performance on challenging cognitive tasks (e.g., Bargh & Cohen, 1978; Burgess & Hokanson, 1964; Hembree, 1988). Furthermore, because individuals generally find it easier to identify linguistic targets presented to the RVF/LH (e.g., Banich, 1997; Rasmussen & Milner, 1977b), some participants in the present study may have shifted their gaze (consciously or unconsciously) slightly to the right (despite explicit instructions to maintain their fixation to where the centrally presented fixation cross was displayed) in an attempt to improve their accuracy and decrease the number of incorrect response feedback messages that they received. If several of the participants shifted their gaze to the right, this would help to explain not only the very

poor accuracy rate for LVF/RH trials, but would also explain the very large VF main effect (whereby participants' accuracy rates for RVF/LH trials was much greater than their accuracy for LVF/RH trials) that was several magnitudes greater than is typically observed with the DVF task (e.g., Banich, 1997). By shifting their gaze to the right, this would have improved their accuracy for RVF/LH trials while negatively impacting their performance on LVF/RH trials. Moreover, if many of the participants were shifting their gaze on multiple trials, overall findings associated with their performance on this task would need to be interpreted with extreme caution because many of the targets were no longer being projected to the intended hemisphere. In other words, if their gaze was shifted to the right, then they were no longer processing those right-sided targets with their left hemisphere exclusively and these targets were being projected to both hemispheres; and left-sided targets weren't being projected to either hemisphere because they were no longer in the visual field. Thus, any findings associated with the VF/hemispheres would be un-interpretable because VF of target presentation (according the computer) could no longer be assumed to relate to processing capabilities within a specific hemisphere (i.e., it's impossible to tell which hemisphere(s) is processing the targets).

Another potentially viable explanation for the poor LVF/RH accuracy rate derives from the present study's list of verbal stimuli. Unlike previous investigations (Atchely et al., 2003, 2005), the present study specifically excluded potential stimulus words that were characterized as either *high arousal* or *low arousal* based on previous norming research (Bradley & Lang, 1999); instead, it employed only *moderately*

arousing words. The rationale behind this choice was the desire to control for the potential effect of stimulus arousal level and, thereby, to better isolate the effect of word *valence* on verbal processing in the study experimental task. However, Atchley and colleagues (2005) have observed significantly higher overall accuracy in judging high-arousal negative words (relative to negative words that are less arousing), and also greater accuracy in judging low-arousal positive words (relative to positive words that are higher in arousal) – a pattern that appears to be lateralized to the right hemisphere. Thus, elimination of the most salient words from the wordlists – that were previously shown to be especially salient for the RH - may have inadvertently compromised overall RH performance on the valence-judgment task.

Lastly, it is important to note that the present study findings suggest that LVF/RH accuracy rates were more seriously compromised for previously-depressed than never-depressed participants; an assertion that is supported by the relatively greater numbers of previously depressed participants who were excluded from subsequent analyses because of poor LVF/RH accuracy rates (17 of 27 previously depressed participants were excluded relative to 8 of 21 never-depressed participants). Thus, in addition to addressing the poor LVF/RH accuracy rates across diagnostic groups, it is also important to address why accuracy judgments were compromised to a greater extent for previously depressed participants in the present study.

One possible explanation for the more seriously compromised DVF task performance of previously depressed participants in the present study relates to the

significantly greater state-anxiety that they reported relative to mood-primed never depressed participants (i.e., previously depressed participants had significantly higher scores on the STAI-State measure). Although, STAI-state scores were not found to significantly influence participants' performance on the DVF task when this variable was included in the omnibus ANOVA model, findings associated with this analysis were difficult to interpret because of the abnormally large VF main effect, which may have been a consequence (at least in part) of multiple participants performing the task incorrectly (i.e., shifting the eyes right, multiple eye movements). Thus, any findings associated with the original omnibus analysis (including null findings) should be interpreted with extreme caution. Moreover, it remains possible that there is a subtle - and negative effect - of state-anxiety on DVF task performance, but the present study design was not sufficiently powerful to detect it. As a result, it is probably best to interpret the non-significant findings for state-anxiety on DVF task performance in the present study as insufficient evidence supporting its influence on this task that warrants further investigation. Moreover, given the challenging nature of the DVF task and the well-documented relationship between elevated levels of arousal (e.g., state-anxiety) and compromised performance on challenging tasks (e.g., Bargh & Cohen; Burgess & Hokanson; Hembree), greater state-anxiety in the previously depressed participants appears to be an alternative explanation for their relatively poor performance (although admittedly lacking in empirical support in the present study) that may facilitate reconciliation within a theoretical framework. Furthermore, as was suggested earlier, feedback given during the DVF experiment (e.g., incorrect

response) may have been stressful for participants, and experienced as particularly stressful for participants who reported feeling more anxious (i.e., reported higher state anxiety) to begin with. Thus, greater state-anxiety experienced by previously depressed participants during the DVF task may also help to explain their inferior performance on LVF/RH trials relative to never-depressed participants.

Another potential explanation for previously depressed participants' compromised performance on the DVF task in the present study relates to the possibility that they may have been more affected by the mood induction relative to never depressed participants (at least initially), and their greater global negative affect (e.g., anxiety and/or hostility according to the MAACL), in turn, may have had a detrimental influence on their performance during the task. Although findings from the VAS do not support this assertion – because there were no significant differences between the diagnostic groups on this measure – findings from the MAACL suggest that the mood induction was initially more potent for previously depressed participants. Moreover, if previously depressed participants were experiencing greater negative affect relative to never-depressed participants during portions of the DVF task, then they may have felt more stressed by the negative feedback (i.e., incorrect response) at those times, and subsequently less confident in their ability to accurately identify targets, which compromised their performance (especially for the more challenging LVF targets). Thus, greater negative affect in previously depressed participants – relative to never depressed participants – during portions of the DVF task may have negatively impacted their performance at these times, and resulted in

their lower accuracy rates (for LVF trials in particular) overall for the experiment. Notably, although it would be difficult to explain their compromised performance for the entire experiment based on this account - because they only reported greater global negative affect (e.g., greater anxiety and/or hostility on the MAACL) during part of the experiment – this interpretation in combination with other factors (including the aforementioned hypothesis) may help to explain their lower accuracy rates relative to never-depressed participants (i.e., a combination of factors contributed to their poorer performance). Furthermore, previously depressed participants reported experiencing greater state-anxiety overall (higher scores on the STAI-state measure that was given at the end of the experiment) relative to never-depressed participants, and therefore, may have been more vulnerable to stressful aspects associated with the DVF task (particularly the more challenging LVF targets).

Finally, an alternative explanation for previously depressed participants' lower accuracy rates in the present study may best be described as sample characteristics of this group that were not measured, but nonetheless differed significantly from previously depressed individuals recruited in other studies, and negatively impacted their performance on the DVF task. For example, two variables that were not measured in the present study, but that would theoretically play a very significant role in performance on the DVF task, is participant motivation and participant distractibility. Motivation and distractibility are participant variables that are particularly significant to studies employing DVF paradigms because the task is challenging and requires sustained attention and motivation to accurately identify

targets presented to each hemisphere/VF. Moreover, diminished motivation or distractibility would be more likely to negatively influence accuracy judgments for targets projected to the LVF/RH, because as was discussed earlier, RVF/LH linguistic targets are generally easier for most right-handed participants to identify relative to LVF linguistic targets (e.g., Banich, 1977; Rasmussen & Milner, 1977b). Thus, if many of the individuals that comprised the previously-depressed diagnostic group in the present study were less motivated or more distracted when performing the task compared to individuals that comprised the never-depressed group, performance of the former group would likely be significantly compromised relative to the performance of the later group. Notably, many of the previously depressed participants were recruited early on in the recruitment phase of the study, and recruitment began in the month of November shortly before the holiday season. Thus, it remains possible that many of the previously depressed participants were more distracted by thoughts of final exams and returning home for the holidays relative to the never-depressed participants, and therefore, were more distracted and/or less motivated to exert significant effort on a challenging cognitive task in the context of a university psychology experiment. Although, this explanation is impossible to support empirically with the present study (because these variables weren't measured), it may help to make some sense of this unexpected finding.

One finding from the present study that was not explicitly predicted – but not entirely surprising - is the observed processing bias for valence-primed negative words across both diagnostic groups (this finding was supported by a significant

interaction between valence and prime-target relationship found with the ANOVA model that excluded participants with poor RH accuracy). Thus, negative target words preceded by a related prime word were judged more accurately than negative targets preceded by an unrelated prime word. There was not, however, a significant valence-priming advantage for positive targets, although the pattern of results for positive targets was consistent with a valence-priming advantage that would likely have been significant if the present study had more power.

There are a number of viable explanations for the greater valence-priming advantage for negative words found in the present study. Notably, for example, this finding is consistent with an extensive literature that documents the heightened overall salience of negatively toned information (e.g., Smith, Larsen, Chartrand, Cacioppo, Katafiasz, & Moran, 2006). For example, research from non-clinical populations (e.g., individuals without a DSM-IV diagnosable disorder) has found evidence of a negative bias in the evaluations that people make (e.g., Kahneman & Tversky, 1984), information that people voluntarily attend to (e.g., Fiske, 1980; Graziano, Brothen, & Berscheid, 1980), and automatic, pre-conscious attentional processes (e.g., Hansen & Hansen, 1988; Ohman, Lundqvist, & Esteves, 2001; Pratto & John, 1991). Furthermore, several researchers have argued that this processing bias with respect to negative information is very robust – i.e., that in many respects *bad is stronger than good* (e.g., Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). One theoretical explanation that seems particularly compelling is the fact that natural selection pressures over the eons gave preference to individuals who weighted

negative information from the environment more heavily than positive information. By this account, the adverse consequences of failing to attend to and accurately process negative information (e.g., the presence of nearby predators) were greater than those involved in the failing to process positive information (the presence of a nearby food supply), such that those who accorded heightened salience to negatively toned information were more likely to survive, reproduce, and pass their genetic material on to subsequent generations. Thus, after thousands of years of natural selection processes, human beings have evolved to more readily attend to negatively valent information in their environment (e.g., Cacioppo et al, 1997). By this account, then, the present finding of a significantly greater priming advantage for negative words across diagnostic groups is not surprising, and might have been expected.

In addition to addressing the unanticipated findings associated with participants' divided visual field task performance (i.e., previous study findings were not replicated), it is also important to address the findings associated with the mood-induction procedure used in the present study. In short, there were no significant effects of the mood induction on any study variables of interest. Notably, however, this procedure may not have produced a persistent sad mood state for either diagnostic group, inasmuch as final VAS (dysphoric mood) scores – obtained about 15 minutes following the mood induction – were not significantly elevated above pre-induction baseline scores. Consequently, the present study's non-significant findings for mood-priming on lateralized emotional processes may be a consequence of ineffective mood-priming rather than the absence of an appreciable effect of transient

sad mood states on these processes. Unfortunately, the present study findings are consistent with both interpretations, and therefore, is unable to adequately address this important theoretical question regarding the significance of sad mood states to lateralized emotional processes, particularly in depression-vulnerable individuals.

In regard to the effectiveness of the mood induction procedure for priming sad mood states, there are a couple of issues that warrant further discussion. First, although the VAS scores for the present study suggested that the mood priming procedure was not sufficiently powerful to produce a sad mood state that endured across the last two blocks of DVF trials, the MAACL scores were more consistent with an enduring sad mood state because when mood primed previously-depressed and never-depressed participants were compared to sham induced never-depressed participants immediately following mood priming, and after the last two blocks of DVF trials, both mood primed groups showed significantly higher depression subscale (and residualized subscale) scores relative to sham induced never-depressed participants. One interpretation of these mixed findings is that the two mood measures are tapping different aspects associated with a sad mood state. In this regard, the MAACL assesses the number of negative and positive adjectives that a participant endorses to describe how they feel at that moment, whereas the VAS requires participants to mark how sad or not sad they feel in that moment on a 10 cm line. Therefore, it is possible that the MAACL is tapping into more of the verbal processes (or even ruminative processes) associated with a transient sad mood state. Furthermore, because the MAACL also requires participants to think about positive

adjectives (and not endorsing them), it may also be assessing the lack of positive affect. The VAS, however, may be assessing the participant's rapid assessment of how they generally feel in that moment without tapping verbal processes that may be activated by a sad mood state, or assessing for the absence or presence of positive affect. However, the implications of these possible differences – between the measures - for the present study is difficult to assess because they were not administered at completely overlapping time points, and therefore may also be measuring different aspects of mood state depending on when they were administered (e.g., before mood induction vs. after mood induction).

Furthermore, perhaps a more parsimonious interpretation of the mixed findings associated with these two measures of mood state is that the VAS was better suited to assessing the persistence of a sad mood for the present study simply because it was administered on multiple occasions, and included an assessment prior to the mood induction procedure. Consequently, the VAS provided a baseline assessment for a within-subjects comparison on the effectiveness of the induction procedure, and several post-mood priming assessments, which permitted the detection of a linear decline for the sad mood state. The MAACL, however, was used in a between-subjects comparison with the sham-induced never-depressed participants – as a control comparison - and was administered immediately following mood priming and after the final block of the DVF task. In this regard, the VAS results for the present study suggested that the mood-priming procedure was not an adequate test of the significance of sad mood states on the lateralization of emotional processes, and

therefore, the non-significant findings associated with this manipulation in the present study should be interpreted with extreme caution and warrant further investigation.

Because the VAS findings for the present study were consistent with a relatively rapid decay of the primed sad mood state, possible explanations for this finding, and whether it may generalize to other studies that have employed similar sad mood priming procedures warrants further discussion. First, the present study is one of a limited number of studies that has used repeated (and multiple) assessments of mood state throughout the experiment to determine whether the primed sad mood persists; however, see Beevers & Carver, Ingram and colleagues, (1994), and Segal and colleagues (1999) for other studies that employed mood state assessment procedures that allowed for detection of decayed or deteriorated sad mood priming effects. Although the aforementioned studies (Beevers & Carver; Ingram et al; Segal et al.) provided support for an enduring sad mood, the vast majority of studies have not employed mood state assessment procedures that enable detection of deteriorating mood priming effects. Therefore, in light of the present study's support for a decayed sad mood state with the VAS findings (before completion of the cognitive task), and the fact that most studies have not tested for this possibility, it raises questions as to why the mood induction procedure may not have produced a sufficiently lasting sad mood state in the present study.

For example, was there something about the divided visual field task used in the present study that facilitated recovery from the primed sad mood state, or, even something unusual about the participants that made them resilient to mood priming

effects? In this regard, it is worth pointing out that the task was challenging and required considerable sustained attention. Therefore, one hypothesis is that the task was sufficiently distracting from ruminative thoughts to contribute to a fast recovery from the primed sad mood. Alternatively, there is the possibility that this finding is a reflection of the transient nature of the primed sad mood state associated with frequently employed mood induction procedures that would otherwise have been observed in many of the other studies if they had assessed for decayed mood priming effects. Unfortunately, until additional studies employ mood state assessment methodologies that test for possible decay effects (e.g., repeated assessments, assessment of mood after all cognitive tasks and/or measures have been completed), this question will remain largely unanswered. However, the VAS findings in the present study raise the question as to whether these commonly employed mood priming procedures are sufficiently potent to reliably produce mood states that persist long enough to adequately assess the effect of sad mood states on cognitive processes.

Lastly, findings associated with the full depression subscale of the MAACL suggested that sad mood priming initially produced greater global negative affect in previously depressed participants relative to never-depressed participants (albeit very transient), but not greater depressive affect because once the variance associated with hostility and anxiety was removed (e.g., the residualized depression subscale scores) there were no longer significant differences between the diagnostic groups. Therefore, this study's findings suggest that sad mood priming may produce greater negative affect associated with anxiety and/or hostility in depression-experienced

individuals, but does not produce greater depressive affect in these individuals. This finding is both novel – in that we are not aware of any other studies that have documented this finding with sad mood priming procedures – and interesting because it suggests that sad mood priming may at least initially produce greater negative arousal in depression-experienced individuals, but not greater depressive affect (which is usually associated with less arousal relative to anxiety and hostility). However, given that this finding was both unpredicted and novel, it warrants further investigation.

As previously mentioned, this study was the first to simultaneously examine the role of state anxiety and a primed sad mood on emotional processing in previously-depressed and never-depressed participants using a divided visual field paradigm. In essence, it represents an attempt to clarify the degree to which previous reports of an enduring right-hemispheric depressotypic verbal processing bias might be contingent on the experience of state dysphoria or state anxiety – a question of some significance because major theories (e.g., Beck, 1976) concerning the etiological roles of negatively biased cognition, and the degree to which such cognition is mood-state-dependent, are still being evaluated (e.g., Hedlund & Rude, 1995; Ingram et al., 1994; Miranda & Persons, 1988) Thus, given the significance of the aforementioned questions to better understanding depression vulnerability, and the fact that the present study was unable to address these questions for reasons already discussed, future research that examines the combined contributions of mood state, anxiety, and cognitive vulnerability to depression seems paramount. Moreover,

because there were several limitations associated with the present study, several recommendations are also warranted.

One of the major limitations associated with the present study may in fact be related to the sample size that was retained and included in the primary study analyses. Although the number of participants recruited for each group in the present study was not appreciably smaller than that of previous studies from the same lab using the same divided visual field paradigm (e.g., Atchely et al., 2003, 2005), many participants in the present study demonstrated very poor accuracy on the DVF task (particularly for LVF/RH trials), and therefore, recruitment of much larger sample sizes seems to be necessary given that many participants may need to be excluded from the analyses. Furthermore, given some of the other study limitations (discussed below), the study likely had less power for detecting reliable and statistically significant findings in the first place, and therefore, required additional participants. Thus, null findings associated with the present study should not be accepted and interpreted as indicative of what would be found in studies with larger sample sizes and greater statistical power.

A second major limitation associated with the present study concerns the strategies used to help participants gain proficiency at performing the DVF task. First, although the present study included a practice session (consisting of 30 trials), and 4 practice trials at the beginning of each experimental block, many of the participants continued to demonstrate very poor accuracy rates for LVF/RH targets, and therefore, it appears as though they required additional practice. Furthermore, the

feedback that participants received following each trial may have been stressful (and disheartening over time) for some of the participants (and especially for previously depressed individuals), and inadvertently contributed to their relatively poor accuracy rates, particularly for LVF/RH trials. Thirdly, it was noted previously that some participants in the present study may have shifted their gaze slightly to the right (or even moved their eyes back and forth across the computer screen), and therefore, the strategies used in the present study to discourage eye movements and maintain fixation for the center of the screen (e.g., explicit verbal and visual instructions to maintain focus on the centrally presented fixation cross) were not particularly effective in the present study. Moreover, the use of feedback after each trial as an implicit reminder to avoid eye movements (i.e., participants were informed that multiple incorrect responses were often a consequence of eye movements), may have actually had a detrimental effect on participants' performance. Thus, many of the strategies used in the present study to facilitate participant performance on the DVF task appeared to be ineffective, and in some cases, may have actually compromised performance.

A third limitation of the present study concerns the selection of verbal stimuli used to assess lateralization of emotional processing. Because high- and low-arousal words were excluded from the wordlists – and there was evidence from previous studies that suggested high arousal negative words and low arousal positive words were the most salient for participants – overall accuracy rates, particularly in the RH, may have been inadvertently compromised. Moreover, severely compromised

response accuracy in the RH for many participants led to their subsequent exclusion from further analyses, which significantly diminished the present study's statistical power for detecting significant within and between-group differences, and increased the likelihood for Type II errors. Thus, exclusion of the extreme arousal words – which were the most salient verbal stimuli - from the wordlists may have indirectly contributed to the present study's relatively low statistical power by compromising participants' ability to accurately judge targets, particularly when presented to the LVF/RH.

Another important limitation associated with the present study concerns the mood manipulation and the mixed findings associated with it. Because the MAACL was only administered post-mood induction, no baseline comparison was available for this measure. This limitation was significant because the mood state measures were not consistent in their support for the effectiveness of the mood manipulation, and having a baseline measure for the MAACL would have resolved remaining questions regarding this issue. Moreover, if the findings from the VAS characterized the effectiveness of the mood manipulation, then the mood induction procedure that was used may have limited the ability of the study to examine this important theoretical question regarding the effect of a sad mood state on the lateralization of emotional processing in depression-vulnerable individuals.

Finally, recruitment of university students for all studies, but particularly for studies of clinically significant disorders such as major depression, limits the generalizability of study findings. Therefore, even though all previously depressed

participants met DSM-IV criteria for a prior major depressive episode, they were still likely not fully representative of previously depressed individuals that would be found in other settings. Furthermore, university students generally reflect a limited age range, such that the mode student is in their early 20s, and are different in other significant ways (e.g., education) that make them not entirely representative of the population.

Given the limitations associated with the present study, several recommendations for future investigations in this area are warranted. First, because the present study was likely under-powered for a variety of reasons, *recruiting* larger sample sizes for both participant groups - to compensate for the possibility that many participants may perform poorly on the DVF task - is a very important and relatively easily remedied recommendation. Future studies that are adequately powered with larger sample sizes for both groups, for example, would help to illuminate whether many of the non-significant findings associated with the present study reflect inherent power issues associated with smaller sample sizes, or important contributions to the literature inasmuch as they might suggest that findings observed by previous studies may rely on specific methodologies that have not been explicitly identified previously (e.g., feedback after each trial may compromise performance) .

Another recommendation that is equally important (if not more important) concerns the strategies used to help participants gain proficiency at performing the DVF task. Given that many participants in the present study evidenced very poor accuracy for LVF/RH trials, additional practice with the task – prior to beginning the

experimental sessions – seems paramount. For example, including at least two practice sessions prior to beginning the first experimental block would give participants twice the amount of practice – relative to the present study – and the opportunity to ask knowledgeable questions (based on their experience with the first practice session) and practice a second time before the actual experiment. Conversely, another strategy aimed at increasing participants' comfort with the task would be to require all participants to gain proficiency (i.e., above chance accuracy judgments) at identifying LVF/RH targets before beginning the experimental sessions (although at some point participants who continued to evidence poor LVF/RH accuracy judgments would need to move forward with the actual experiment). Secondly, given the likelihood that the performance feedback is stressful (or least not helpful) for many participants (and this may be particularly true for previously depressed participants), eliminating the feedback after each trial is recommended. Finally, because participants may find it difficult to remain vigilant for eye movements throughout the DVF sessions, having a researcher stay in the room with them while they perform the DVF task, and periodically remind them to continue to fixate their eyes on the center of the screen (where the fixation cross was presented), may help to reduce the number of eye movements produced by participants during the task.

An additional recommendation for future studies is to use verbal stimulus lists that include high- and low-arousal words, because exclusion of these words in the present study may have inadvertently contributed to the relatively poor LVF/RH

accuracy rates that were observed for many participants in the present study. Furthermore, it is recommended that positive and negative words be balanced for the arousal level associated with each, and that equal numbers of low, moderate, and high arousal words for both valences be included. Experimentally manipulating the arousal level of the word stimuli by including equal numbers of high and low arousing words for each valence would allow for the exploration of the singular effect of valence – and the effect of arousal – without eliminating what are likely the most salient words (high arousing negative and low arousing positive words), and thereby reducing power to detect significant differences within subjects (e.g., valence priming advantage for negative and positive words) and between diagnostic groups. Moreover, if the arousal level of verbal stimuli contributes differentially to cognitive biases among the different diagnostic groups, then including verbal stimuli that possess the full range for the arousal dimension permits exploration of such possibilities.

Another recommendation alluded to earlier, involves the assessment of mood priming procedures. Although the present study represented a significant improvement over the vast majority of previous studies that have employed mood priming procedures to investigate the influence of sad mood states on cognitive vulnerability in depression, the omission of a baseline measure of the MAACL prohibited the unequivocal evaluation of the effectiveness of the mood induction procedure. Therefore, it is recommended that future studies use repeated assessments and incorporate multiple measures of mood state as was done in the present study, but

also include a baseline assessment of mood state for all mood state measures that are used. By including baseline assessments of all mood state measures, and repeated assessments of mood state, questions regarding the potency of mood priming procedures may become more conclusive.

Finally, even though there were no observed effects of state anxiety on the lateralization of emotional processing in the present study, given that the study was likely underpowered because many participants were excluded from the analyses, and findings associated with the initial omnibus analysis are likely unreliable and difficult to interpret for reasons already discussed, it remains possible that there were more subtle effects of anxious symptomatology on emotional processing that the present study was unable to detect. Thus, it is recommended that future studies continue to assess the possible role of state anxiety in the lateralization of emotional processing. Moreover, because depression and anxiety disorders are highly comorbid, future studies may also want to assess for these disorders (in addition to major depression) in order to determine whether more severe anxious symptomatology affects information processing biases in depression that are not observed with more transient anxious states.

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